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MITRAL COMMISSUROTOMY PERFORMED FROM THE RIGHT SIDE

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HISTORICAL ASPECTS OF MITRAL COMMISSUROTOMY

THE development of an efficient surgical treatment for mitral stenosis has, like most other significant medical advances, been preceded by a long period of observation and investigation, formulation, and finally clarification of concepts. After an interval of trial and error, significant clinical success has been achieved in a large number of cases. However, there has been no cessation in our efforts to extend the benefit of surgery to a wider group of patients and also qualitatively to improve the individual result. The present report describes a new development which, it is felt, has much to offer. The historical background of this conceptional change is interesting and pertinent.

The first good description of the pathologic entity which we know as mitral stenosis was given by de Vieussens[1] in 1705. In 1819, Laennec[2] correlated the clinical features of the disease with the observed pathologic changes, discussed the probable disturbances of physiology, and described the auscultatory findings which make an accurate diagnosis possible during life. A direct surgical attack upon the stenotic valve in order to enlarge the orifice ("slightly notching the valve") was proposed by Samways,[3] in 1898, and by Brunton,[4] in 1902. The latter specified that the surgical incisions should lengthen the valve slit.

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Cutler's[5] pioneering intervention, in 1923, using a valvulotome and the transventricular route in which he converted a stenotic mitral valve aperture into a larger, although less competent one, set the pattern of surgical attempts for the next twenty-odd years, culminating in the brilliant efforts of Smithy[6] who performed Cutler's procedure on 7 patients with five survivals. However, the considerable amount of regurgitation necessarily produced by these attempts militated against true clinical success and often precluded operative survival.

Souttar,[7] in 1925, on the other hand, successfully "dilated" a stenosed mitral valve by manipulations of the index finger which had been inserted into the left atrial chamber through an incision made in the tip of the left auricular appendage.

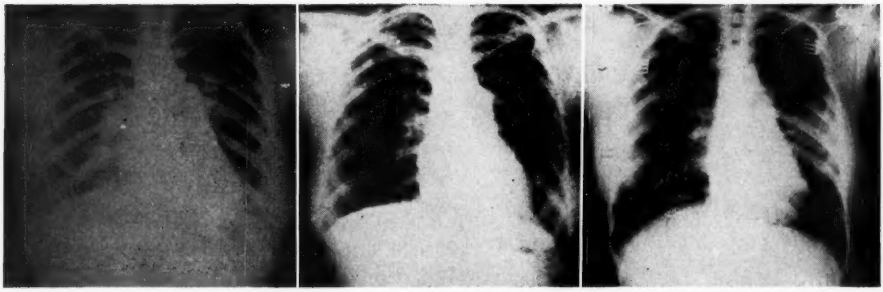


Fig. 1.—Preoperative, six months postoperative, and 6½ years postoperative (left to right), follow-up roentgenograms in C. W., the first patient historically to undergo a modern type of operation for mitral stenosis. (Courtesy Pennsylvania M. J. 53: 6, 1950.)

In 1948, after preliminary unsuccessful clinical attempts dating back to 1945, the senior author presented the concept of separating the fused valve leaflets anatomically along the lines of the obliterated commissures, thus overcoming the valvular obstruction without the inherent necessity of producing any regurgitation. This concept is the basis of the modern corrective operation for mitral stenosis, and has been designated as "commissurotomy."[8] The first successful intervention of this type in a patient with classical mitral stenosis was accomplished on June 10, 1948. A hooked knife attached to the finger was used to divide the valve. The mobilized leaflets, thereby, were rendered capable of function of a normal type; no regurgitation was created. The auscultatory evidences of disease have been nearly abolished, and the heart size has become reduced progressively since operation (Fig. 1). This young woman continues to remain in excellent health to the present time more than eight years after operation.

Independently of the American investigations, in September, 1948, Brock[9] successfully carried out an operative procedure which was based upon a similar concept of mobilizing the fused leaflets, but which was accomplished by splitting with finger pressure alone. A logical intermediate step, although chronologically performed later, between those procedures in which regurgitation necessarily was produced and the modern type of operation, was

Harken's[10] original "valvuloplasty" procedure. On June 16, 1948, he successfully enlarged a stenotic mitral valve orifice by instrumental resection of portions of the mural valve leaflet at the price of what he called "selective insufficiency."

TECHNICAL FEATURES OF MITRAL COMMISSUROTOMY AS PERFORMED FROM THE LEFT SIDE.—From that point, up to the present time, there have been relatively few major changes in the over-all method of commissural separation. The right index finger is passed into the left atrium through an incision made in the tip of the left auricular appendage. Resistant valves are separated at the commissures by instrumental means, while the more amenable are split by manipulations of the intracardiac finger alone. Whenever the subvalvular structures are cross-fused below the valve orifice, as described by Brock,[11] they must be split apart or divided instrumentally as described by Nichols.[12] One major advance has been the introduction of the "bare finger" technique by Husfeldt.[13] This increases the operator's tactile perception and hence accuracy of manipulation by several hundred per cent.

In the majority of the patients who have been operated on up to the present time, only the anterolateral commissure has been separated. The effective reason for this is as follows: The posteromedial commissure seemingly is appreciably more resistive to blunt disruptive pressure as it is applied from the left side, and instrumental attempts at division of this portion of the valve substance (from the left) often are attended by the creation of considerable valvular incompetence.

In approximately 20 per cent of the first 1,200 patients operated on at our Clinic, there was recognized, in addition to the obliteration of the commissures, a significant degree of subvalvular obstruction to blood flow due to a complex interfusion or cross-fusion of the chordopapillary supporting tissues. This actual hindrance to blood flow must be distinguished from clinically insignificant instances of thickening and fusion of contiguous chordae which may be demonstrated to exist in the vast majority of patients with clinical mitral stenosis. In most instances, any secondary obstruction has been overcome readily by direct digital pressure. In some patients, in whom the papillary muscles are directly adherent to the valve apex, instrumental splitting of these muscles, as described by Nichols,[12] may be necessary in order to establish an adequate subvalvular passageway. This was carried out in six instances in the present series. Such mechanical separation, whenever necessary, of subvalvular "adhesions" of the chordopapillary system to permit free separation of the leaflets logically is included within the term "commissurotomy" in the same sense that separation of pleural adhesions and mediastinal dissection, when necessary, are included within the term "pneumonectomy."

Good clinical results have been reported upon sizable series of operative patients by numerous authors.[11, 14-16] However, the periods of follow-up have been relatively short in the over-all course of rheumatic valvular disease and the reported "long-term" benefits have been limited approximately to 65 to 75 per cent of those so treated (Table I).

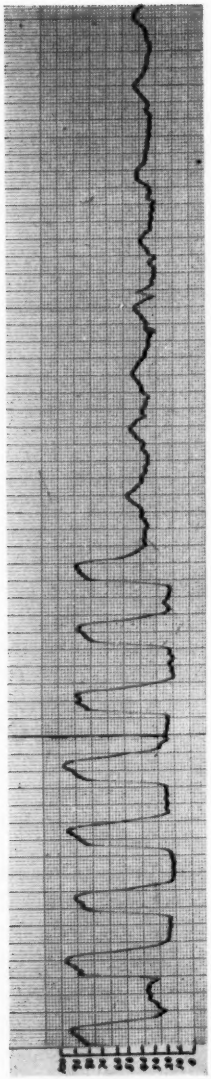


Fig. 2.—Pressure tracing obtained in R. H. during left heart catheterization as catheter was withdrawn from the left ventricle into the left atrium. The gradient of 30 mm. Hg existing across the mitral valve during left ventricular filling indicated a severe state of restenosis of the mitral valve in a woman who had been markedly improved for the first three of four years following mitral commissurotomy. Reoperation from the right side confirmed the diagnosis (8 mm. by 3 mm. valve slit) and overcame the obstruction much more adequately than had been accomplished originally (from the left). (Courtesy H. Goldberg, Brith Sholom Cardiopulmonary Laboratory, Philadelphia, Pa.)

TABLE I. RESULTS IN FIRST 1,000 PATIENTS OPERATED UPON FOR MITRAL STENOSIS—LEFT-SIDED APPROACH (LONGEST FOLLOW-UP—8 YEARS)—AUTHOR'S SERIES

	NUMBER OF CASES	PERCENTAGE
Mitral commissurotomy	1,000	100
Operative deaths (excluding 2 cases of suicide)	77	7.7
Late deaths (2 of these deaths were due to carcinoma)	56	5.8
Follow-up of survivors		
Improved	711	89.8
Unchanged	49	6.2
Worse	32	4.0

Unable to follow up 73 cases.

ANALYSIS OF UNSATISFACTORY RESULTS.—Leaving out of consideration for the moment those patients who do not survive operation (2 to 10 per cent), we find an appreciable number (10 to 15 per cent) who are not helped initially by the procedure, or are even made worse by it. This group includes patients in whom the stenosis has been relieved but slightly or not at all, and also those in whom a significant amount of regurgitation has been produced. It does not include those patients who suffer postoperatively from recognized or unrecognized pleural or pericardial effusion which ultimately resorbs with clinical improvement. A presently indeterminate number of patients exhibit definite and often marked improvement for a variable number of months or years, but gradually redevelop symptoms and signs similar to those which brought them to the cardiac surgeon initially. In such individuals, left heart catheterization nearly always reveals definite evidence of re-establishment of an obstruction to blood flow at the level of the mitral valve—elevation of diastolic gradient between left atrium and ventricle (Fig. 2).

A frequently unsuspected but relatively common cause of an unsatisfactory clinical result is unrecognized or incompletely appreciated coexistent disease of another valve—either the aortic or tricuspid. That such rheumatic multivalvular involvement is not uncommon is clearly shown in Table II. It will be noted in this table that in the clinical studies tricuspid disease was recognized rarely, if at all, while its incidence in the autopsy studies ranged from 10 to 25 per cent.

Recurrent Mitral Stenosis.—Brock,[17] Jordan and Hellems,[18] Keyes and Lam,[19] and others have reported cases in which they have attributed clinical regression to restenosis of the valve. In some, the diagnosis has been confirmed by reoperation and direct palpation of the valve. In others, the opinion has been based upon subsequent autopsy findings. It is our belief that, with the increasing number of mitral interventions and the passage of time, such reports will become more frequent in the near future.

Harken and Black,[20] and Glover and his associates[21] have objected strongly to the concept of recurrence of mitral stenosis, believing restenosis of the surgically well-opened valve to be extremely improbable. They feel that such instances of re-establishment of obstruction at the mitral level are due basically either to incomplete opening of the fused commissures at the original operation or to failure

TABLE II. INCIDENCE OF VALVULAR HEART DISEASE*

	NEW ENGLAND		VIENNA	BERLIN	JOHNS HOPKINS, BALTIMORE
	CABOT	WHITE	KAUFMANN	SPERLING	HIRSCHFELDER
	208 CASES POST-MORTEM (%)	1,097 CASES CLINICAL (%)	126 CASES POST-MORTEM (%)	300 CASES POST-MORTEM (%)	1,781 CASES CLINICAL (%)
Mitral	85.6	56.3	60	85	51
Aortic	44.7	14.7	76	43	22
Tricuspid	15.9		25	10	
Pulmonary	1.9			1	
Aortic and mitral	19.2	28.9			20
Aortic, mitral, and tricuspid	11				
Mitral and tricuspid	2.9				
Pulmonary and tricuspid	1				
All four valves	1				

*From Paul D. White, M.D., Heart Disease, ed. 4, New York, The Macmillan Publishing Company.

of the operator to relieve a subvalvular obstruction. We agree that such incomplete surgery predisposes to early secondary narrowing of the valve passageway and must be a major factor in the re-establishment of mitral obstruction. However, this very re-establishment of the stenosis after initial partial relief must, by definition, be admitted to constitute *recurrence* of the obstruction.

Indeed, we feel the precept, which one of us (C. P. B.) formerly advanced,[8] that wide opening of but a single commissure constitutes an adequate surgical mobilization of the valve in most cases, has done much harm. Similarly, Harken's[10] strongly promulgated dictum (which he has since abandoned[22]) that an adequate splitting of the valve may be accomplished nearly always by digital pressure alone (instrumentation, therefore, rarely being necessary), has been equally unfortunate. It is now clearly evident to both proponents of these erroneous concepts that the widest feasible mobilization of both obliterated commissures is indicated and that an instrumental attack is necessary to accomplish this in at least one-half of all operative patients. Undoubtedly, these early misconceptions have vitiated, in certain respects, the value of the statistical follow-up upon both these sizable operative series. Furthermore, these precepts, having been accepted by many other surgeons as sound, have redounded to the disadvantage of their patients as well as themselves. No doubt many patients so treated will become candidates ultimately for more adequate reoperation.

Recurrent Stenosis After Complete Mobilization of the Valve.—There remains a group of patients with indubitable recurrent stenosis of the adequately opened and mobilized valve. The occurrence of this condition can be established only if a surgeon of considerable experience in this field carefully reports the initial operative result and then, months or years later, personally reoperates on this patient or observes the valve at autopsy.

We are able to report fourteen instances of proved recurrent stenosis in patients who had had unicommissural mobilization of the valve. We also have

reoperated on 2 patients in whom complete bipolar commissurotomy had been accomplished previously and in whom extensive refusion of the commissures had taken place. These are being reported in detail elsewhere. Probably there have been many unrecognized cases of this condition among the great number of patients who have been subjected to mitral valve surgery throughout the various clinics in the world, since the possibility of its occurrence has not been widely appreciated. Doubtless some of these individuals, even now, erroneously are presumed to be suffering from irreversible myocardial or pulmonary vascular disease.

The extreme pathologic changes which have taken place in many of the valves of our operative patients must tend to reproduce or to perpetuate the tendency of the process to produce complete obliteration of the orifice. Slowly moving, stiffened valves, even though divided well beyond the scarred margin of the passageway, may remain "hinged" upon partially fibrosed tissue. Further, it would seem that the surgically produced irregularities of the cut valve surfaces, as well as those produced by the existing pathologic alterations, must continue to produce the flow turbulence to which Magarey[23] attributes the recurrent surface deposition of fibrinous exudate which appears to be the basic element in the pathogenesis of mitral stenosis. In all probability, once the valve pathology has become advanced sufficiently, the stenosing process, even though set back mechanically by one or more successful surgical interventions, inevitably must continue to progress as long as life exists. In this respect, a very severe stenotic deformation of the mitral valve apparently behaves like the fibrous stricture which it really is.

On the other hand, a mitral obstruction, which may be just as tight mechanically as the preceding type, may be found unassociated with extreme pathologic change in the leaflets. Extensive but light cross-adherence of the leaflets' margins has greatly reduced the size of the valve aperture. It would appear improbable that these flexible and mobile cusps would become re-fused extensively following adequate surgical separation unless the patient were to suffer from another bout of rheumatic fever.

While in valves with the extremes of pathology, recurrence of mitral stenosis would seem to be inevitable, regardless of the surgical technique employed, and while in others with minimally diseased leaflets, re-fusion following separation would seem to be extremely unlikely, there is a large intermediate group of patients. In them, it is probable that the adequacy of surgical valve mobilization largely determines the likelihood and rate of re-establishment of the obstruction at the mitral level.

Certainly it would seem logical that, following a previous commissural separation, the unquestionably rheumatic patient would be likely to redevelop a stenotic valvular lesion in the event that he should suffer another bout of rheumatic "activity." And, in any case, whether due to specific exacerbation or to residual mechanical irregularities of the valve surfaces, it seems reasonable to assume that this admitted tendency toward restenosis must be greater with respect to the valve leaflets than to the subvalvular supporting structures.

This order of likelihood would seem to be implicit in the designation of such subvalvular changes as "secondary stenosis," the actual leaflet lesions causing "primary stenosis."

Whatever the actual incidence of recurrent stenosis following adequate commissurotomy may prove to be eventually, it is probable that most instances of re-establishment of obstruction at the mitral valve up to the present time have been favored by incomplete surgery, commissural separation having been extended insufficiently or an element of coexisting subvalvular stenosis having been overlooked or ignored. All experienced cardiac surgeons, in retrospect, can recall instances in which they have failed in these respects.

Inadequate Surgical Experience.—Tragically, as yet, there appears to be little organized effort on the part of the profession to prevent further errors of this type from being made by beginning cardiac surgeons in the future. A young and enthusiastic surgeon with grossly inadequate training in the field of cardiac surgery may not hesitate to insert an index finger into the left atrium with the intention of performing mitral commissurotomy, even though, as an extreme example, he may never have performed any major cardiovascular operation previously. Repeatedly, the senior author (C. P. B.) has been astonished to meet surgeons who never have obliterated a patent ductus arteriosus or resected a coarctation of the aorta, but who claim to have done a number of "mitrals." It is extremely unlikely that they have completely split the valves in 50 per cent of the cases which may be reasonably amenable to digital separation of the commissures. Certainly they have not effectively treated the 50 per cent which require instrumentation because they have used no instrument. More likely, each has swept a finger around the atrial chamber in a state of complete disorientation and then has removed the digit triumphantly from the heart without even palpating the valve orifice. Since the finger and valve cannot be seen, who is to know what really has transpired within the heart? Usually, with modern anesthesiologic and cardiologic control, the patient will survive and the case will be listed, again triumphantly, as a successful mitral commissurotomy operation. With the intense psychologic impression which such an operation creates, and with the interested and more concentrated medical attention which then is accorded him, the patient may feel much improved during the next few months. Ultimately, as real improvement fails to materialize and the disease progresses sufficiently, he will deteriorate perhaps to be listed as a case of "recurrent stenosis." This tragic situation is reminiscent of that which persisted for some time after the introduction of the endoscopic transurethral method of prostatic resection.

Technical Difficulty of the Operation.—Such amateurish surgical attempts already have brought about a considerable amount of discredit to the operation of commissurotomy.[24, 25] Yet, experience and ordinary common sense indicate that this operation is a highly technical one, frequently requiring skillful use of special instruments under the sometimes unfamiliar guidance of

"digital vision." Often, full valvular mobilization is extremely difficult to accomplish. Further, the operation inherently is associated with dangers of causation of cardiac arrest, arterial embolization, and mitral regurgitation. To permit even a naturally skillful and generally well-trained surgeon who happens to be unfamiliar with this technique to attempt such an operation, which must be performed by the guidance of the sense of touch alone, is a reflection upon our medical intelligence and integrity. If it takes the accepted minimum of 50 transurethral resections, properly performed under supervision, to prepare a novitiate for the practice of that phase of the specialty of urology,[26] it would seem that at least an equal number of episodes of training must constitute the minimum preparation acceptable for the practice of the surgical treatment of mitral stenosis.

Dissatisfaction With Mitral Commissurotomy Operations as Usually Practiced.—While the operation of mitral commissurotomy performed from the left side has been becoming gradually more acceptable to the profession and even has achieved high favor in some circles, the shortcomings of the standard technique have become increasingly obvious to your authors. For a long time, no satisfactory solution to these problems has seemed to be within reach. Difficulties experienced in simultaneously treating coexisting rheumatic lesions of the aortic and tricuspid valves and the irreducible incidence of arterial embolization occurring during mitral surgery have increased this dissatisfaction, although our major complaint has been related to the unsatisfactory technical accomplishments.

THE RIGHT-SIDED APPROACH

In a letter to Sir Horace Mann, dated Jan. 28, 1754, Horace Walpole[27] said that he invented the term "Serendipidy" on the title of a fairy tale, "The Three Princes of Serendip," because the princes were "always making discoveries by accident and the sagacity of things they were not in quest of." In the same sense the discovery of the advantages of performing mitral commissurotomy from the right side surely must be accredited to Serendipidy, since it was incidental to attempts to perform simultaneous surgery for right-sided noncardiac lesions.

In 1954, the first description of an approach to the mitral valve from the right side, with a report upon 3 clinical patients, was presented by Neptune and Bailey.[28] At that time, it was felt that the indications for such an approach in mitral stenosis were relatively few, consisting mainly of the coexistence of right-sided extracardiac lesions amenable to surgery, or of definitely diagnosed multivalvular stenosis (aortic or tricuspid) which would require a right thoracic intervention. Since the submission of that report, further advances in the development of the technique and a better understanding of the utility of this approach have caused us gradually to adopt it as our routine method for the performance of mitral commissurotomy in the absence of appropriate contraindications. Some of the advantages and disadvantages of the right-sided operation are listed in Table III.

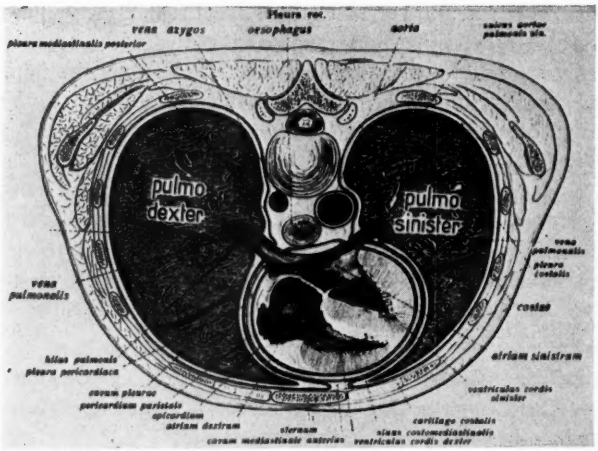


Fig. 3.—Diagrammatic representation of relationships of cardiac structures at the level of the mitral valve. Note that the left atrial chamber extends fully as far toward the right side as toward the left, and that it logically might be called the "posterior" atrium. (Courtesy Sobatta, München, 1920, J. F. Lehmanns Verlag.)

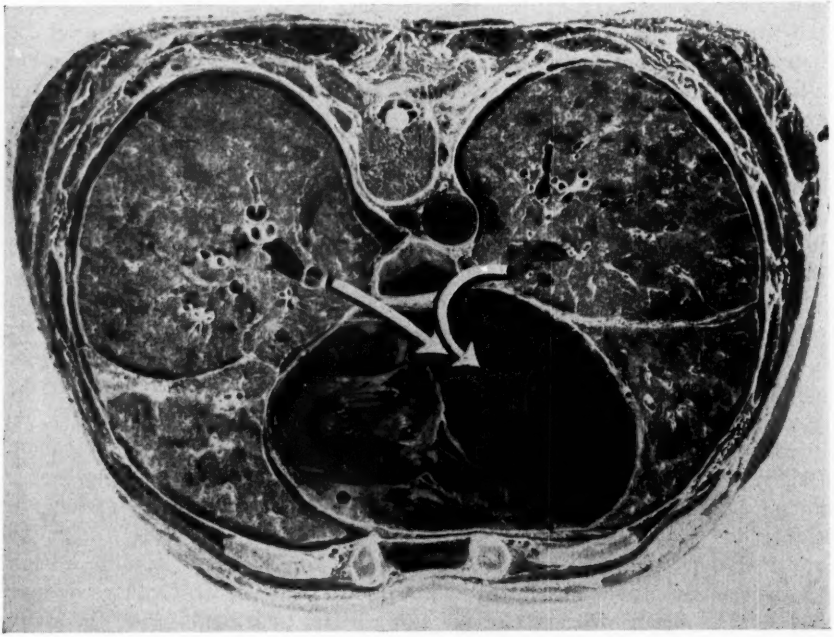


Fig. 4.—Actual cross section of human thorax at the level of the mitral valve. Note that while the mitral valve orifice actually does lie somewhat to the left of the midline, the approach to it by way of the left auricular appendage (see arrow) is basically an awkward one in that the finger after insertion must practically be doubled back upon itself to reach the commissures. In contrast (see arrow), the right-sided approach brings the finger tip to the valve in direct end-on fashion which greatly facilitates all manipulations. (Courtesy P. Seara, Estudio Anatomico Topografico Del Torax y Del Abdomen, Buenos Aires, Argentina, 1941).

TABLE III, A. ADVANTAGES OF THE RIGHT-SIDED APPROACH

1. A superior mitral commissurotomy may be accomplished with full and safe mobilization of both commissures
2. Unsuspected and marked coexistent tricuspid disease may be diagnosed readily and treated appropriately
3. Coexistent aortic stenosis may be relieved by the preferred transaortic technique through the same operative incision
4. Operative arterial embolization due to manipulative dislodgment of thrombotic material from the atrial appendage may be avoided
5. The supine position is physiologically easier on the patient
6. A coexistent unsuspected interatrial septal defect may be diagnosed digitally and treated at the same operative session
7. Concomitant right-sided noncardiac disease may be treated simultaneously
8. Conditions involving the left thorax may render left thoracic cardiotomy more difficult or dangerous; this refers particularly to previous left-sided mitral intervention, also thrombotic obliteration of the appendage, and the rare condition of calcification of the left atrial wall
9. The incision is shorter and less traumatic, and causes less postoperative pain and shoulder dysfunction
10. The skin incision averages 27 cm. in length compared to 47 cm. with the left posterolateral incision, and is hidden largely within the inframammary fold in the female patient (cosmetic advantage)

TABLE III, B. DISADVANTAGES OF THE RIGHT-SIDED APPROACH

1. The pertinent anatomic orientation is entirely different from that of the left-sided approach, and must be learned before the commissures can be mobilized accurately and fully
2. For the surgeon inexperienced in this approach, hemostasis is technically more difficult to achieve because there is no appendage upon the right side of the left atrium
3. Pressure on the right side of the heart may produce severe although transient hypotension
4. It is more difficult to extract a loose thrombus from the left atrium from the right side
5. The left auricular appendage cannot be amputated in an effort to prevent recurrent arterial embolization; however, this amputation is believed now to be unnecessary with the relief of stasis afforded by satisfactory enlargement of the valve
6. Cardiac massage, if necessary, cannot be carried out effectively from the right side in most instances
7. Should an unexpectedly large amount of mitral insufficiency be encountered, the appropriate corrective measures cannot be applied from the right side (see Fig. 6, *A*, *B*, *C*)

An objection has been raised to the performance of mitral commissurotomy through a right-sided incision on the basis that it is "an operation for a left-sided lesion performed from the right." Yet, this same critic[29] very recently has recommended the performance of tricuspid commissurotomy (truly a right-sided lesion) through a left thoracic approach!!

In reality, as shown in Fig. 3, the left atrium extends anatomically just as far toward the right side as toward the left. It more properly might be designated at the posterior atrium. Actually, the mitral valve orifice does lie slightly to the left of the midline, much as the entire heart is directed somewhat to the left (Fig. 4). However, this minor anatomic deflection is of slight importance in comparison with the many technical and pathologic features which favor the right-sided route for mitral commissurotomy.

This report is based upon our experience with 200 patients who were operated on by the right-sided approach for significant mitral stenosis, with

or without coexistent aortic or tricuspid disease. The first patient was operated upon on Nov. 1, 1954, and the last on Oct. 8, 1955.

ADVANTAGES OF THE RIGHT-SIDED APPROACH.—

An Improved Commissurotomy.—While originally the performance of commissurotomy from the right side was described[28] as technically more difficult than that through the usual left auricular appendage approach, and while, at first, our various staff surgeons* felt insecure and less than normally competent while attempting to separate the fused valve commissures from this new and "unusual" approach, subsequent experience has brought about improved orientation and greatly has increased our technical skill in the various manipulations entailed in the performance of commissurotomy from the right side. Today, we are of the unanimous opinion that a markedly superior mobilization of valvular structures may be achieved from the right than usually is feasible from the left. The term "mobilization" is used advisedly. By it we mean, not only, the anatomic separation or splitting apart of the fused leaflets, but also, the restoration of leaflet function by separating any cross-fused subvalvular supports.

In contrast to the general experience that it is difficult to open the posteromedial commissure from the left side, when the mitral valve is approached from the right, this commissure lies conveniently in line with and just below the site of cardiac entrance and may be opened accurately and safely by digital or instrumental technique in nearly every instance. This valve extremity has been opened in all but 3 of our first 200 patients operated on from the right side. Such separation of the posteromedial commissure rarely is attended by the creation of significant regurgitation. It is somewhat more difficult to separate the anterolateral valve commissure by digital pressure alone from the right side, partly because of the difficulty in applying external counterpressure. However, instrumental division is found to be no more difficult than when using the left lateral approach and either instrumental or digital opening of the fused anterolateral commissure was also accomplished in all but 2 of the 200 cases. Table IV shows the reason why the other commissure was not opened in the 5 cases in this series in which only one was attacked. The directions of approach to the valve orifice from the respective two sides are contrasted in Fig. 4.

TABLE IV. THE REASON FOR NOT OPENING A COMMISSURE IN THE 5 CASES WITH ONLY ONE COMMISSURE OPENED

H. M.	loosely attached calcium in posteromedial commissure—feared brain embolus. Anterolateral opened
E. B.	eccentric orifice extending anteriorly to the valve ring; posteromedial commissure opened completely
C. H.	chordae tendineae adherent only to one border of posteromedial commissure; feared insufficiency. Anterolateral opened
L. A.	posteromedial commissure heavily calcified; no fusion of leaflets anteriorly; posteromedial commissure opened
H. M.	heavily calcified and regurgitant posteromedial commissure; feared production of increased regurgitation if separated. Anterolateral opened

*Charles P. Bailey, Houck E. Bolton, Henry T. Nichols.

The above statistics are compared with our experience in the first, 1,200 patients with mitral stenosis upon whom we operated from the left side (Table V). In them, bicommissural separation was accomplished in 33.1 per cent. The anterolateral commissure alone was separated in 65 per cent. The posteromedial alone was divided in 1.9 per cent. It is our considered opinion that the average size of the opening achieved when the operation is performed from the right side ranges from 33 per cent to 300 per cent larger than that which may be expected with the left-sided approach (Table VI). This additional enlargement of the orifice is not attended by any increase in the incidence of significant mitral regurgitation which might be created or magnified by the surgery.

TABLE V. NUMBER OF COMMISSURES OPENED FROM THE RIGHT-SIDED APPROACH COMPARED WITH THE LEFT-SIDED APPROACH

	LEFT-SIDED APPROACH (1,200 CASES) (%)	RIGHT-SIDED APPROACH (196 CASES*) (%)
Both commissures opened	33.1	97.3
One commissure only		
Anterior	65.0	1.6
Posterior	1.9	1.1
Total	100	100

*Four cases operative data not complete.

TABLE VI. VALVE SIZE ATTAINED IN MITRAL COMMISSUROTOMY FROM THE RIGHT SIDE AS COMPARED WITH THE RESULTS FROM THE LEFT

	RIGHT SIDE		LEFT SIDE	
FINAL VALVE SIZE	NUMBER OF CASES	PER CENT	NUMBER OF CASES	PER CENT
Less than 1½ finger opening (up to 2.4 cm.²)	5*	2.5	23	2
1½ finger opening (2.5 cm.²)	56	28.0	549	46
2 finger opening (4 cm.²)	133	66.5	519	43
Over 2 finger opening (4 cm.² plus)	6	3.0	30	2
Unknown	0	0	79	7
Total	200	100	1200	100

*Three of these were table deaths with the valve surgery not completed.

Subvalvular stenosis: We have observed no reduction in the facility with which subvalvular chordopapillary fusion masses may be divided. If unseparated, they may prevent otherwise adequately mobilized valve leaflets from separating during diastole. If they are sufficiently extensive, they may provide a secondary site of atrioventricular obstruction below the level of the actual valve orifice. In 29 of the 200 patients with mitral stenosis operated upon from the right side, thickening and cross-fusion of the chordopapillary supports was recognized and relieved; in 6 by definitive instrumentation. Although in most instances such secondary obstruction can be overcome readily by direct digital pressure, there are some patients in whom the papillary muscles may be directly adherent to the valve apex and instrumental splitting of these muscles, as described by Nichols,[12] may be necessary in order to establish an adequate subvalvular passageway.

A.

B.

C.

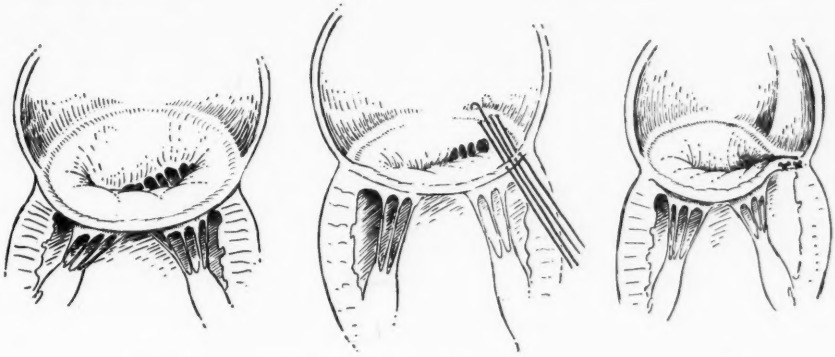
Fig. 5.—*A*, Marked thickening and partial fusion of adjacent chordae which does not cause significant subvalvular obstruction and which may be seen in many stenotic rheumatic mitral valves.

B, Extensive subvalvular chordal change and cross-fusion which does substantially reinforce the obstruction produced by the primary cross-adherence of the leaflets. (Courtesy Bailey: Surgery of the Heart, Lea & Febiger, p. 509.)

C, Extreme subvalvular change with direct union of agglomerate chordopapillary pillar-like masses with the apex of the valve funnel which has become elongated by the organization of fibrinous exudate deposited upon the free margins of the thickened leaflets. Here the subvalvular pathology is extreme and will require definitive separation, perhaps by instrumental means, in order to relieve the circulatory obstruction. (Courtesy Surg. Clin. North America, 32: 1,807, 1952.)

It must be realized that while minor pathologic changes may be found in the chordae of most patients with mitral stenosis, and while various degrees of agglutination or clumping of adjacent chordae supporting the same leaflet are seen frequently, cross-fusion of the chordae to the opposite leaflets in such a form as to cause a true subvalvular stenosis is relatively less common (Figs. 5, *A, B, C.*).

Mitral insufficiency: Should an element of mitral incompetence (perhaps previously entirely unsuspected) be found to exist, it should be evaluated as to amount and to site of localization with respect to the leaflet structure and the extremities of the line of valve closure. This information along with an appraisal of the condition of the leaflets (calcification, retraction, etc.) and the supporting chordopapillary system will furnish the basis for a decision as to whether a later operation[30] through a left-sided incision may be indicated (Fig. 6, *A, B, C*).



A. *B.* *C.*

Fig. 6.—Nichols' method of polar cross-fusion of the annulus fibrosus for the correction of mitral insufficiency. (Courtesy New York J. Med., March 15, 1956.)

A, Typical posteriorly incompetent mitral valve with retraction of free margin of the posterior portion of mural leaflet. A similarly localized area of incompetence may be noted in cases of extreme annular dilatation.

B, Plicating sutures placed about or through apposed portions of the annulus fibrosus across the incompetent pole of the valve, but preferably not actually over the valve aperture.

C, Tying down these sutures brings the selected portions of the annulus fibrosus into intimate early and, by healing, permanent contact. This reduces the over-all size of the area bounded by the annular ring, and by bringing the attached basal portions of the retracted leaflets closer together permits their free margins to come into full approximation during ventricular systole.

MULTIVALVULAR INVOLVEMENT

The predominant tendency toward localization of the chronic rheumatic valvular lesions, as indicated in Table II, evidently is upon the mitral valve, but aortic involvement is a reasonable second, and tricuspid affection is far from infrequent. Apparently, the tricuspid valve never is found to be stenotic in the absence of a similar lesion of the mitral valve. However, isolated disease of either the aortic or mitral valve is seen not uncommonly.

Although the pulmonary valve, early in the course of rheumatic disease, exhibits classical rheumatic microscopic lesions involving the annulus fibrosus, it almost never develops the late features of leaflet thickening and commissural obliteration which are so characteristic of the rheumatic process.

It must be recognized, of course, that considerable pathologic alteration of a valve must take place before a clinical effect is recognizable. Minor degrees of leaflet thickening and beginning commissural fusion may produce auscultatory changes long before significant effects are imparted to the circulation. For severe disturbances of physiology to develop more profound pathologic changes necessarily must take place.

Aortic Stenosis.—The logical approach to correction of a stenotic lesion of the aortic valve has been shown to be[31-34] by way of a pouch sutured to the ascending aorta, using a thoracic incision made through the right third or fourth interspace, anteriorly. It must be appreciated that the newer methods of evaluation of aortic stenosis (left heart catheterization; preferably combined simultaneous catheterization of the right side of the heart to determine the cardiac output by the Fick principle and of the left side of the heart to determine the pressure gradient across the valve) permit a rather exact preoperative determination of the amount and degree of physiologic obstruction of this valve which may be present. Hence, it should rarely be considered necessary to explore the aortic valve digitally for the purpose of making an exact diagnosis. The preoperatively determined systolic gradient which exists across this valve gives one the essential criterion upon which to base a decision for or against definitive intervention. It is felt that a systolic gradient of 40 mm. Hg across the aortic valve (or more), in isolated aortic stenosis, is necessary to justify aortic exploration.

Aggravation of the Effects of Aortic Stenosis Following Mitral Commissurotomy.—It is realized that in certain cases of combined mitral and aortic stenosis, the obstruction at the level of the mitral valve may be so severe that the relatively small amount of blood which passes through it readily can traverse even a fairly small aortic valve orifice without creating a significant pressure gradient across this valve. In such an individual, surgical relief of the mitral obstruction may produce such an increase in the blood flow through the aortic valve that a much larger pressure gradient (systolic) may develop across it, postoperatively. Direct measurement of this aortic gradient at operation before and after the mitral valve has been opened has not proved to be completely satisfactory, although it is employed in all doubtful cases. Apparently, this is due to a reduction in the cardiac output due to anesthesia, thoracotomy, opening of the pericardium, and operative blood loss (Fig. 7, *A*, *B*). This may be considered a "physiologic damping" of the pressure curves. However, failure to relieve a severe coexisting aortic stenosis at the same time that a mitral obstruction is corrected constitutes a major disservice to the patient since isolated aortic stenosis runs a more rapid devolutionary course than does combined mitral and aortic stenosis of equal severity. In our early experience, we have had several patients in whom a mild thrill over the aorta, at the time of surgery, was adjudged insufficiently pronounced to indicate aortic surgery. A few of these patients not only failed to improve following mitral commissurotomy, but went on later to die of their aortic disease.

Except in the case of those relatively few patients with combined aortic and mitral stenosis who present an equivocal or no pressure gradient across the aortic valve, we believe that the surgeon who undertakes to operate for dynamically significant aortic stenosis logically should attack this valve before the mitral, using an appropriate thoracic incision (now nearly always the right third anterior interspace). Having completed the aortic surgery up to

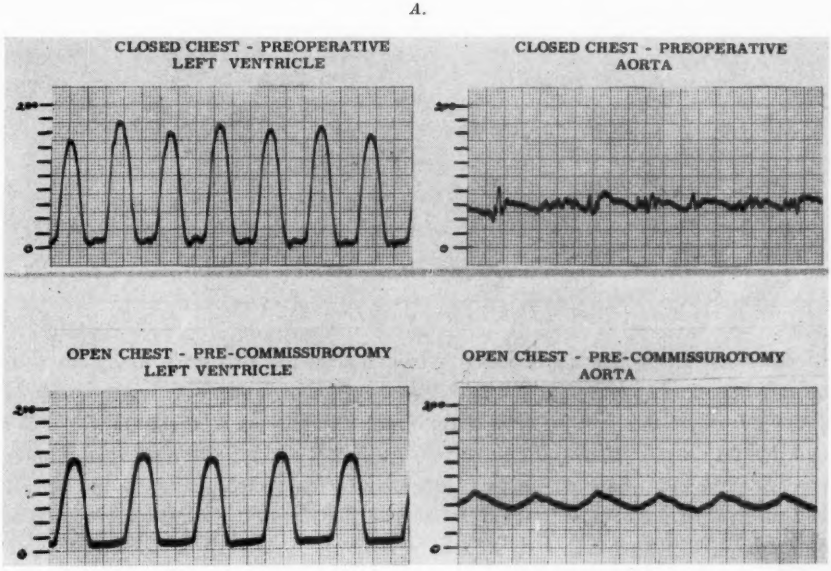


Fig. 7.—"Physiological damping" of the pressure curves obtained by direct needle puncture respectively within the aorta and the left ventricle during the course of surgery. (Courtesy H. Goldberg, Brith Sholom Cardiopulmonary Laboratory, Philadelphia, Pa.)

A, Pressure curves obtained preoperatively by left heart catheterization in Patient W. R. with severe aortic stenosis. Note the large systolic gradient across the aortic valve.

B, Similar tracings obtained in the same patient by direct puncture of the aorta and left ventricle at the time of operation prior to the definitive aortic commissurotomy. Note the damping of the curves and the reduction in the gradient.

and including the suture repair of the aortic wall, he then will explore the mitral valve digitally if there is any physiologic (catheterization), auscultatory, or other evidence (such as atrial fibrillation) which is suggestive of mitral valve disease.

Tricuspid Stenosis.—In contrast to the situation which pertains with combined aortic and mitral stenosis in which at least the proper diagnosis nearly always may be made from the characteristic physical and physiologic findings, it should be emphasized that serious degrees of tricuspid stenosis or regurgitation frequently may remain unsuspected even after extensive clinical and physiologic investigation; the effects of these lesions being largely obscured

by the more obvious manifestations of the ever-present associated mitral disease. Although Reale, and his co-workers[35] have described well the physiologic findings which are characteristic of its presence, in practice, significant tricuspid disease can be recognized with certainty only by direct digital exploration of the tricuspid valve at time of mitral surgery (Table VII, A).

TABLE VII, A. DEGREE OF TRICUSPID STENOSIS FOUND IN 175* CASES OF TRICUSPID EXPLORATION AMONG 200 CONSECUTIVE MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

		TOTAL	PER CENT
Severe Stenosis		11	6.2
Finger-tip opening	3		
1 finger opening (1.5 cm.²)	8		
Moderate Stenosis		8	4.5
1¼ finger opening	3		
1½ finger opening (2.5 cm.²)	5		
Minimal Stenosis		21	12.0
1¾ finger opening	3		
2 finger opening	10		
2½ finger opening	4		
Unspecified minimal involvement	4		
Total		40	22.7

*In 25 patients tricuspid exploration was not carried out.

Exploration of Tricuspid Valve.—Insertion of the operator's finger through an incision made in the right auricular appendage to explore the right atrial chamber when using a left-sided approach is difficult and dangerous. We employed it in 7 patients, the first time on Jan. 23, 1953, and described it only to condemn it.[16] Any attempt at instrumentation of this valve from the left is fraught with unusual danger due to the poor exposure and control of the appendage. In truth, this is an attempt to perform a right-sided operation through a left-sided approach!! Tricuspid commissurotomy performed through a right thoracic incision, on the other hand, is readily, efficiently, and safely accomplished. If we are to include a routine exploration of the tricuspid valve with every operation for mitral stenosis, the approach must be made from the right.

Among the 200 patients, 30 were operated on primarily for aortic lesions. There were 170 patients who were operated on for a diagnosed mitral stenosis, although in 2 an additional preoperative diagnosis of tricuspid regurgitation, and in 5 of tricuspid stenosis, had been made. These suspected additional valvular lesions were found, and also another fourteen instances of significant (moderate to severe) tricuspid stenosis (Table VII, A), as well as another 7 cases of previously unsuspected significant incompetency of the tricuspid valve (Table VII, B) in 175 of these patients in whom the tricuspid valve was explored. In 25, hypotension or other untoward signs precluded elective examination of this valve. Had we failed to perform digital examination of the interior of the right atrium in the majority of this group of patients, the multivalvular involvement in most cases would have escaped notice and in

many of the individual patients would have contributed to an unsatisfactory or to an incompletely satisfactory clinical result. These considerations have impressed us with the necessity for routine digital exploration of the tricuspid valve in every patient who is operated for mitral stenosis, provided there is no contraindication.

TABLE VII, B. DEGREE OF TRICUSPID INSUFFICIENCY OCCURRING IN 175 CASES OF TRICUSPID EXPLORATION AMONG 200 CONSECUTIVE MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

	NUMBER OF CASES
Not Associated With Stenosis	
Severe insufficiency* (3 plus)	3
Moderate insufficiency (2 plus)	3
Minimal insufficiency (1 plus)	14
With Stenosis (Before Tricuspid Commissurotomy)	
Severe insufficiency (3 plus)	0
Moderate insufficiency† (2 plus)	3
Minimal insufficiency† (1 plus)	11
Total	34

*Tricuspid annulus fibrosus plicated in 2 patients.
†Included in Table VII, A.

TRICUSPID REGURGITATION.—Tricuspid insufficiency, fortunately, is an abnormality which physiologically is less difficult to bear than are equivalent regurgitant lesions of the other two valves which are subject to rheumatic deformation (aortic and mitral). Nevertheless, in its more severe forms, it is a distinct burden to the patient, especially when it is found in one with increased pulmonary arteriolar resistance, a condition often associated with (and caused by) mitral stenosis. With such increased vascular resistance, effective function of the right ventricle becomes especially important in order to propel the blood through the pulmonary vascular bed. This is accomplished with great difficulty in the presence of a seriously incompetent tricuspid valve since during systole the right ventricular content more easily may be delivered in retrograde fashion through this valve into the systemic venous bed rather than into the pulmonary artery.

The correction of tricuspid insufficiency by direct, digitally controlled plication of the external portion of the tricuspid annulus fibrosus, as previously described,[36] may be carried out expeditiously and almost without significant additional risk through the right anterior thoracic approach (Fig. 8, *A-E*). However, it can be performed only with difficulty if the incision has been made at a level higher than the right fourth interspace (the fifth is preferable). In 2 of these patients, such plication effectively corrected the regurgitation. We believe this procedure to be indicated whenever the regurgitation is estimated (digitally) to be of a magnitude greater than 2 plus, providing, of course, that the patient's general condition is deemed satisfactory for such additional surgery. Recently, it has been possible to overcome severe isolated tricuspid insufficiency in one patient (D. C.) by the Nichols'[30] method of polar cross-plication of the anterolateral portion of the tricuspid annulus fibrosus (Fig. 9, *A-C*).

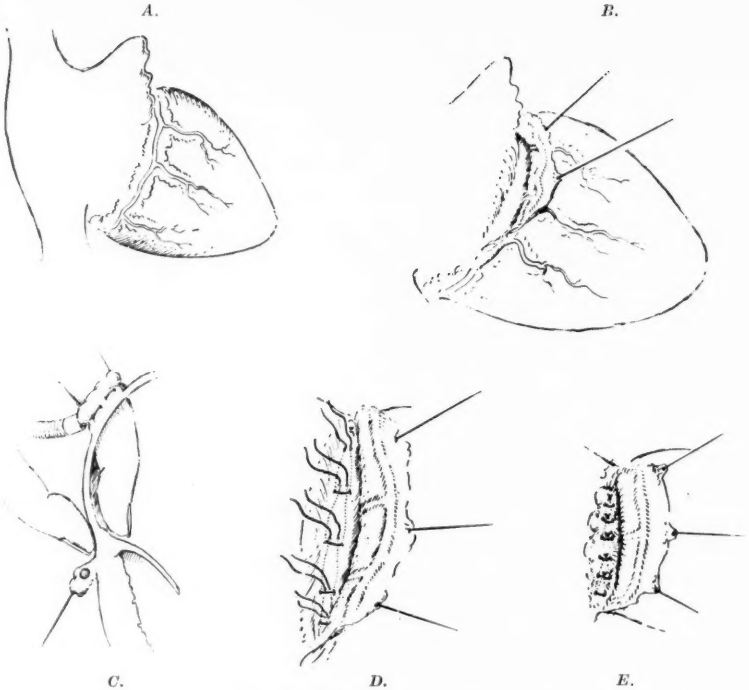


Fig. 8.—Partial circumferential plication of the mural portion of the tricuspid annulus fibrosus for insufficiency of this valve. (Courtesy Bailey, et al., Cardiovascular Surgery, Proceedings, Henry Ford Hospital Symposium, 1955, W. B. Saunders Company.)

A, Right atrioventricular sulcus.

B, Mobilization of fat pad and contained right circumflex coronary artery from the sulcus.

C, Left index finger inserted into right atrium through incision made in right atrial appendage and thumb placed beneath dissected fat pad (and right circumflex coronary artery) to palpate annular ring.

D, Placement of interlocking heavy mattress sutures into and about a portion of the perimeter of the valve ring.

E, Tying down of these sutures effectively reduces the length of this portion of the ring.

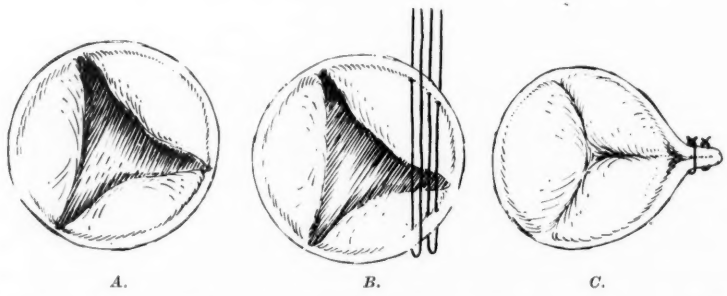


Fig. 9.—Nichols' method of polar cross-plication of the annulus fibrosus applied in tricuspid regurgitation.

A, Preoperative condition of the valve with either a dilated ring or shrunken leaflets resulting in a significant disproportion between the available existing leaflets substance and the size of the aperture to be defended.

B, Placement of plicating sutures in the annulus over the anterolateral extremity of the triangular valve orifice.

C, Incompetence of the valve is markedly reduced or overcome as the sutures are tied down and the bases of two of the leaflets are brought closer together.

Appendageal Thrombosis.—Since dislodgment of loosely attached thrombotic material from the appendage (atrial thrombosis was present in 29 per cent of our first 1,200 patients with mitral stenosis in whom commissurotomy was performed from the left side) during the entrance of the operating finger or during the subsequent intracardiac manipulations is believed to be the chief and usual cause of systemic arterial embolization when it occurs during surgery (Table VIII), one logically might expect to eliminate such accidents from this source by choosing a different site of atrial entrance. Using the dissected interatrial groove as the approach, the entrance is made through the left side of the interatrial septum, a site characteristically free of thrombotic attachment. As previously reported,[16, 37] with the routine application of transient constriction to the carotid vessels, when the left atrial lumen is entered via the left auricular appendage, the incidence of cerebral embolization occurring at the time of surgery can be reduced from 5.1 per cent to 1.8 per cent. However, since this technique merely redirects the course of any dislodged particulate matter into the descending aorta rather than through the carotid arteries, the incidence of peripheral arterial embolization becomes increased correspondingly.[16]

True Atrial Thrombosis.—Large extensions of thrombosis from the left auricular appendage into the true atrial lumen were recognized in 3.9 per cent of our first 1,200 patients (operated on from the left). In most instances, the clot forms in "placenta-like" fashion, coming partially to line the left or posterior wall of the left atrium. Usually such "lining" clots are found in an organized state and are well attached.

However, in some patients, massive clotting occupying up to one half or more of the volume of the left atrial chamber may be encountered. If the operator's finger, after recognizing this situation, is kept carefully away from such (usually soft) masses, apparently there is little danger of dislodgment of a portion.

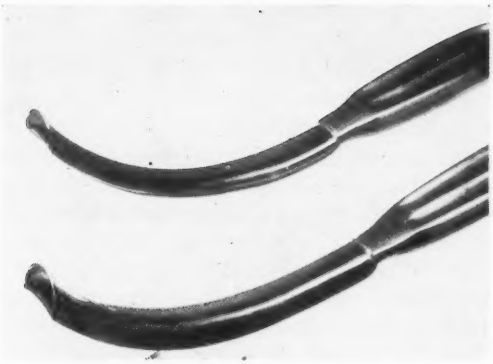
Extensive intra-atrial thrombosis was recognized at surgery in 16 of our 200 patients (8 per cent) operated upon from the right side.

Occasionally, the operator may recognize the presence of a soft freely floating thrombotic mass within the left atrial chamber. In still others, a similar mass may be found to be attached loosely, perhaps by a thin pedicle, and capable of moving freely within a wide arc. Either of these types of "loose" thrombus may lead to the "ball valve" syndrome with intermittent total blockage of the diminutive mitral valve orifice.[16, 38] On March 21, 1955, in Patient L. G. (Table VIII), a soft, loosely attached clot, measuring approximately 3 cm. in diameter, became detached during the surgical manipulations incident to enlargement of the valve aperture, and thereafter disappeared from the left atrial chamber. Immediate palpation of both femoral arteries revealed disappearance of pulsations. Upon completion of the thoracic surgery, an abdominal incision was made and the embolic mass was removed from the aortic lumen close to its bifurcation. Recovery was uneventful.

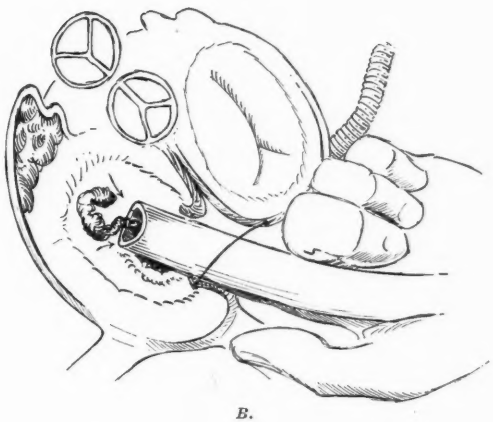
Such poorly attached or floating clots sometimes may be evacuated from the momentarily opened heart by a technique of permitting the pressure of

TABLE VIII. OPERATIVE EMBOLIZATION AMONG THE FIRST 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

PATIENT	PROCEDURE	CLOT IN ATRIUM	CALCIFICATION IN LEAFLETS	TO	OUTCOME
P. R.	Mitral comm., aortic explored	Large, soft granular	Posterior commissure and septal leaflet	Brain	Survived with alexia and agraphia only
L. G.	Mitral comm., tricuspid comm.	3 cm. in diameter, above ant. comm.	Moderate	Aortic bifurcation	Survived; removed at same operation; no complications
M. L.	Mitral re-operation, tricuspid explored	Large, soft	Slight	Midbrain	Survived; diplopia only; improving
W. B.	Mitral comm., tricuspid explored	Numerous small vegetation-like clots	Moderate	Right femoral artery	Survived; complete relief with con. caudal block only; 3 days
M. M.	Mitral comm.	None	Embolus believed of calcific origin; perivalvular nodular calcification located diffusely around all edges of both leaflets and commissures; felt to be loose during surgery	Brain	Died 24 hours; spastic biplegia; no recovery



A.



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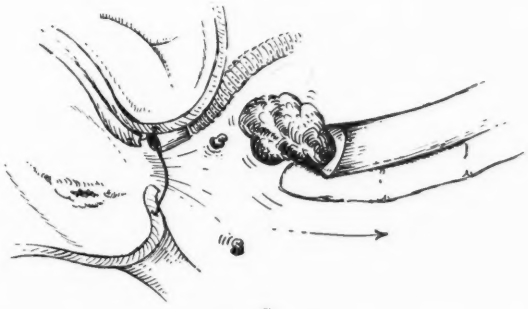


C.

Fig. 10.—Use of one of the Litwak* aspirating tips to remove a loosely attached or freely floating thrombus from the left atrial chamber (right-sided approach).

A, Photograph showing two sizes of the instrument. (Courtesy New York J. Med., March 15, 1956.)

B, Introduction of streamlined instrument along the palmar surface of the intracardiac finger from the right (it is used similarly from the left). The suction is applied after the tip of the aspirator has been brought into contact with the clot. (Courtesy New York J. Med., March 15, 1956.)

C, Sudden removal of the finger, the instrument, and the attached clot from the heart, *en masse*. The concomitant momentary free bleeding from the heart wound serves to "flush out" any smaller fragments which may have become detached during the extraction.

*Obtainable from George P. Pilling & Son Co., Philadelphia, Pa.

the left atrial blood content to force it out, as described by Jamison,[39] and others. However, this maneuver may fail, especially if the mass is large or partially adherent to the endocardial surface.

In such a case, the end of an appropriately shaped, specially designed aspirating tip such as that devised by Litwak (Figs. 10, *A, B, C*) may be inserted into the atrium along with the finger until the end is in contact with the clot. The suction then is turned on and the instrument with attached clot (and finger) is withdrawn through the atrial site of entrance as the purse-string suture is relaxed momentarily. We have performed this maneuver successfully from the right side on three occasions.

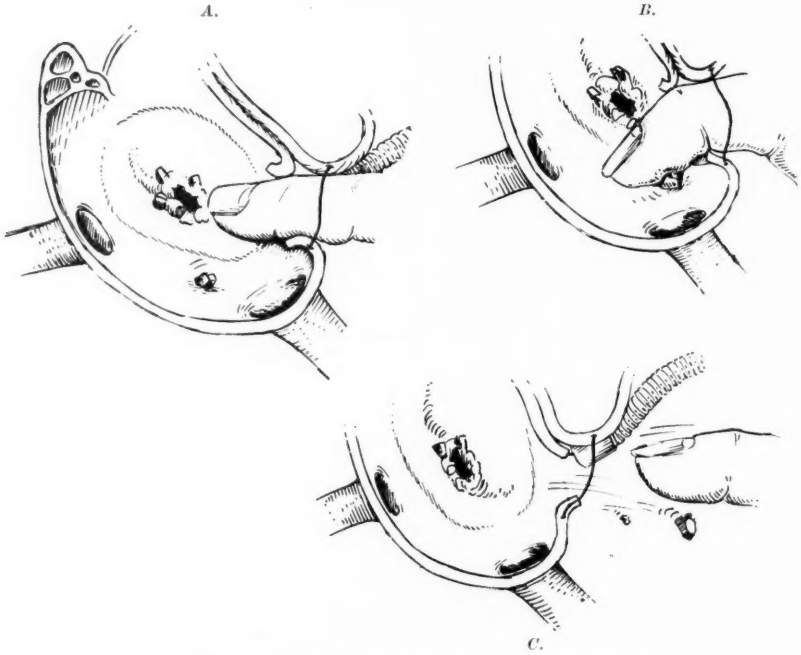


Fig. 11.—Extraction of dislodged calcific particles from the left atrium.

A, Calcific fragments being heavier than blood, when detached from the atrial aspect of the mitral valve by the surgeon's finger or instrument, tend to fall into and to rest for a while upon the most dependent wall of the left atrium.

B, When found by the bare index finger tip of the surgeon, such a particle may be held firmly against the internal surface of the atrium and then "slid" along it to the site of digital entrance.

C, The finger and particle together are removed suddenly from the heart, permitting momentary external bleeding. This tends to "flush out" any overlooked additional particulate matter. Hemostasis is re-established promptly by tightening the purse-string suture.

Calcification of the Valve as a Source of Emboli.—Naturally, fragmentation of calcific material which may be attached to the valve edges is not influenced by the choice of the site of entrance into the left atrium but only by the condition of the valve and by the manipulations entailed in the particular commissurotomy technique employed. Since such dislodged calcific particles are heavier than the blood, they may not be expelled into the

aorta immediately but may come to rest temporarily upon the inner surface of the most dependent wall of the left ventricle or atrium. From the latter site, they may be extracted by sliding them over to the site of atrial entrance with the ungloved index finger[16] (Fig. 11, *A, B, C*).

As shown in Table VIII, all but one of our five instances of operatively produced embolization were of thrombotic origin.

SURGERY FOR "RECURRENT" MITRAL STENOSIS

Obviously, a ready surgical approach from the right side would avoid many of the technical difficulties inherently associated with operation for mitral stenosis for the second time from the left. In contrast to a nearly virginal operative field on the right (except for pericardial adhesions), the left side then is encumbered by the presence of pleuropulmonary adhesions which tend to be very firm over the site of the previous pleural and pericardial incisions. Not only does the pericardium tend to be densely adherent to the stump of the partially amputated appendage, but the latter usually will be found to have become shrunken and imbedded within dense fibrous connective tissue. The important anatomic markings which normally delineate the limits of safe entry into the left atrial chamber are obscured. Hence, in these individuals the right-sided approach to the mitral valve may be made safely and methodically, while the left is fraught with unusual technical problems and the danger of causing air leaks and prolonged oozing from the scarred pleural surfaces, as well as increased real and fancied difficulties in separating the valve commissures. Not infrequently an unsatisfactory valve mobilization may result from hasty manipulations necessitated by insecure hemostatic control during secondary procedures carried out by the left-sided route.

CASE REPORT

F. B. (Case No. 36918), a 23-year-old white woman was operated upon by one of us (C. P. B.) for mitral stenosis at the Hahnemann Hospital, in Philadelphia, on Feb. 27, 1950. She was the fifteenth patient operated on at this hospital for this condition. The approach was made through an opening in the tip of the left auricular appendage, using the usual long posterolateral thoracic incision and opening the fourth intercostal space widely. The double glove technique then in vogue was employed.

The valve was found to be fibrosed but not calcified, and the orifice was contracted to a tiny slitlike aperture less than a centimeter in length and perhaps one fourth of that distance in width. There was no detectable regurgitation (Fig. 12, *A*).

The screw-type guillotine knife was applied against the anterolateral valve commissure and it was cut once. This separation was extended readily and fully to the annulus fibrosus by digital pressure without producing any regurgitation. Palpation with the finger tip revealed adequate support with numerous chordae attaching to both leaflets. It was estimated that the valve opening had been extended more than 1 cm. beyond the line of direct tendinous support.[17]

The operator's attention was turned toward the posteromedial commissure and digital pressure was made against it. At first it resisted strongly. The guillotine knife was closed once. Digital pressure was applied and suddenly the gloved finger seemed to break through the valve substance, separating the tissues widely. The resulting opening surely would have accommodated 3 fingers simultaneously. Immediately, a 4 plus grade of regurgitation became apparent, although palpation of the posterior portion of the valve opening revealed that there were several chordae attached to each lip of the valvular wound. The

posterior separation had become extended at least 2 cm. beyond the posterior line of direct tendinous support and fully to the annulus fibrosus. Presumably, it extended well beyond all normal chordal support and, therefore, even though apparently made in the proper anatomic line, permitted regurgitation due to reflux of the nonsupported portion of the flexible valve edges (Fig. 12, *B*).

The patient's heart dilated enormously, became visibly weaker in its contractions, and went into arrest. The finger was removed from the atrium, the appendage was ligated, and cardiac massage was commenced. With the administration of adrenalin and continued rhythmic compression of the ventricles, the heartbeat returned and a peripheral pulse and blood pressure became obtainable.

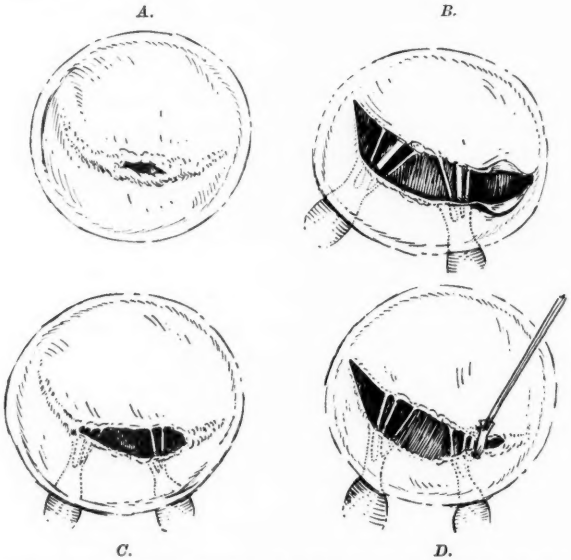


Fig. 12.—Diagrammatic representation of mitral valve in patient (F. B.) demonstrating the significant degree of restenosis which occurred within a five-year period following extensive bicommissural mobilization of the structures.

A, Original stenotic valve orifice in a fibrosed but largely flexible valve.

B, Following an extensive commissurotomy at each valve extremity, a very severe posteriorly located regurgitant jet was noted (estimated to be of 4 plus magnitude).

C, Five years later, at reoperation, the anterolateral commissure had become obliterated to the anterior line of direct tendinous support, and the posteromedial had become refused partially. The regurgitation had become reduced significantly.

D, Direct suturing together of the leaflets over the incompetent pole of the valve reduced the regurgitant jet. The anterolateral commissure had been reopened.

The chest wall was closed with some difficulty because of the great dilatation of the left ventricle which scarcely could be fitted within the thoracic cage. It was impossible to approximate the edges of the widely opened pericardium so the sac was left entirely open.

While the patient remained hypotensive for days, eventually she made a reasonably satisfactory recovery, although the heart showed marked enlargement and there was an entirely new 4 plus systolic murmur over the apex. The previously present diastolic murmur was entirely gone. Atrial fibrillation persisted.

During the next four years, the patient continued to present evidence of mitral insufficiency with a lowered myocardial reserve, tendency to edema of the lower extremities, and engorgement of the liver.

On April 3, 1955, this patient was readmitted to the Hahnemann Hospital (Case No. 55-04017) for repair of the mitral insufficiency. Cardiac enlargement was 3 plus and the left atrium was so enlarged as to be considered aneurysmal.

Operation was performed on April 23, 1955. Again the approach was made through the left thorax, the old incisional scar being opened. Great difficulty was experienced in mobilizing the left lung from the thoracic wall and the myocardium to which it was extensively adherent. Numerous small lacerations of the lung were produced with resulting air leaks.

Even before entering the extremely dilated left atrium, ventricular fibrillation developed once. Spontaneous rhythm was reestablished by massage, electric countershock, and the administration of vasopressive drugs. Nevertheless, the blood pressure remained at a low level. Consultation with the family resulted in a decision to proceed with the surgery in view of her generally downhill course and the improbability of survival from a nonhelpful exploratory operation. Four subsequent episodes of ventricular fibrillation punctuated the ensuing operative procedure, each requiring electric countershock.

A purse-string suture was placed in the atrial wall at the base of the largely obliterated appendage and a stab incision was made within its confines. The operator's bare index finger was inserted into the atrium and the valve was examined. A 2½ to 3 plus regurgitation was present, but a remarkable change had taken place within the valve structures. The anterior commissure had become readherent all the way back to the anterior line of direct tendinous support. Posteriorly, the valve separation no longer extended to the annulus fibrosus, but scarcely a centimeter beyond the posterior line of direct tendinous support which could be delineated clearly with the bare finger. The entire valve aperture was considered to be a 1½ finger size. The obliterated anterior commissural tissue now was definitely calcified (Fig. 12, *C*).

At this point, ventricular fibrillation occurred again, requiring massage and stimulation.

The anterior commissural tissues were split readily to the valve ring without altering the existing regurgitation.

Ventricular fibrillation recurred, requiring electric countershock and massage for its correction.

Using the Jamison stitcher, a strip of pericardium was passed in suture fashion through puncture wounds made in both leaflets at points just beyond (posterior to) the posterior line of direct tendinous support. This technique has been described previously.[16, 40] One end of the pericardial loop was passed through an "eye" made in the other, and the end of the loop thus created was drawn out of the atrial incision. Moderate traction upon this loop significantly diminished the regurgitant jet, leaving an effective valve opening more than sufficient to admit 2 fingers (Fig. 12, *D*).

The procedure was terminated by suspending the end of the pericardial loop from the atrial incision which was closed with cross-suturing of a mattress type. The chest wall was closed in the usual manner with water-seal drainage.

Ventricular fibrillation ensued twice during the closure of the chest wall.

The patient's blood pressure remained at a very low level (60 to 70 mm. Hg, systolic) in spite of transfusions and vasopressive agents, and she failed to react from the anesthetic. Twelve hours after the completion of the operation, the heartbeat disappeared.

Post-mortem examination confirmed the operative findings plus evidence of pulmonary edema and old infarcts of the spleen and left kidney.

Comment.—The clear-cut digital evidence of extensive re-establishment of commissural fusion both anterolaterally and posteromedially in this case, following a more than adequate bicommissural mobilization of the leaflets, leaves no room for doubt that mitral restenosis sometimes will take place despite the most extensive technical surgical mobilization of the valve.

COMPLICATING PLEUROPULMONARY LESIONS

To less extent, the mentioned difficulties may apply in patients who previously have had left-sided thoracic surgery for some other reason, and in those

who have suffered, on a former occasion, from left pleural effusion or empyema, or perhaps even from extensive (left) pulmonary disease such as bronchiectasis, abscess, or tuberculosis. The right-sided approach to the mitral valve makes it possible to by-pass such difficulties in some cases, and with other individuals to undertake simultaneous corrective surgery when indicated for right-sided noncardiac associated lesions.[28] This applies especially to decortication of organized exudate which may form upon the right lung when it has been compressed over a long period of time by pleural effusion which may have been of cardiac origin (failure).

INTERATRIAL SEPTAL DEFECT

We have encountered one small, previously undiagnosed, interatrial septal defect which we were able to close by the technique of atrio-septo-pexy at the time of the right-sided operation for mitral stenosis, as well as two others which were diagnosed correctly, preoperatively. In these latter instances, the mitral valve was approached by way of the right auricular appendage and the septal defect.[41, 42]

ANOMALOUS PULMONARY VENOUS DRAINAGE

Right-sided anomalies of the pulmonary venous drainage may be diagnosed and treated appropriately during the course of the routine exploration of the right atrium. In one of the patients, with interatrial septal defect referred to above, an associated anomalous right pulmonary vein was transposed with the help of the left index finger inserted within the right atrial chamber by a technique described previously.[16, 18]

INCISIONAL PAIN

One of the lesser but nevertheless worth-while advantages of the right-sided thoracic incision is the relative mildness of the thoracic pain experienced during the postoperative period. This was dramatically illustrated in the reports of each of the 6 patients who had secondary surgery performed from the right after previously undergoing left-sided mitral surgery. It must be noted that this minimization of discomfort is equally prominent when the left anterolateral thoracic incision is employed for mitral commissurotomy and merely represents the inherent superiority of the anterolateral over the posterolateral thoracic incisions in this respect.

ARM AND SHOULDER FUNCTION

In part, the prolonged limitation and stiffness of movements of the shoulder girdle which sometimes follow surgical procedures which utilize posterolateral thoracic incisions are related to the initial operative pain and consequent muscle spasm. The voluntary immobilization of these structures which often is established during the immediate postoperative period, may lead to adhesion formation about the shoulder joint, to bursitis, or to a habit of maintained chronic muscular tension which tends permanently to inhibit free movement of the shoulder girdle. A high-held or frozen shoulder and

even a degree of scoliosis may follow. While early and persistent attention to movement tends greatly to diminish the incidence of these complications, every thoracic surgeon has had experience with at least a few individuals in whom these efforts have failed, a permanently stiff and painful shoulder resulting.

In striking contrast, such shoulder complications are almost never seen when an anterolateral thoracic incision is employed.

DIVISION OF THORACIC MUSCLE TRUNKS

The anterolateral incision divides relatively few fibers of the pectoralis major muscle. By a splitting technique, the fibers of the lower third of the muscle mass are separated anatomically over the interspace selected for thoracic entrance, but their innervation, which comes from the axillary region by way of the middle and lateral anterior thoracic nerves, is not interrupted. Hence, the function of this muscle ultimately becomes restored nearly completely to normal.

In dismal contrast, the usual long posterolateral thoracic incision necessarily divides the continuity of the latissimus dorsi completely. At the same time, it denervates the caudal portion of this muscle permanently since it entails division of the spinal accessory nerve filaments which run over and through the muscle structure roughly parallel with the course of its fibers. The rhomboidei and the serratus magnus muscles suffer serious but less extensive damage.

It would seem inevitable that a significant portion of the muscular strength of the shoulder and arm must be destroyed by this type of incision. While such a reduction in the structural strength of the arm, shoulder, and trunk may be of little consequence in a person of sedentary habits, in one accustomed to greater physical activity and vigor, it may reduce in value the benefit obtained from a successful attack upon the valve by precluding a return to his previous vocation or avocations.

What part this weakening of the muscular support may play in the postoperative development of a stiff shoulder has not been determined.

COSMETIC ASPECTS OF THE INCISION

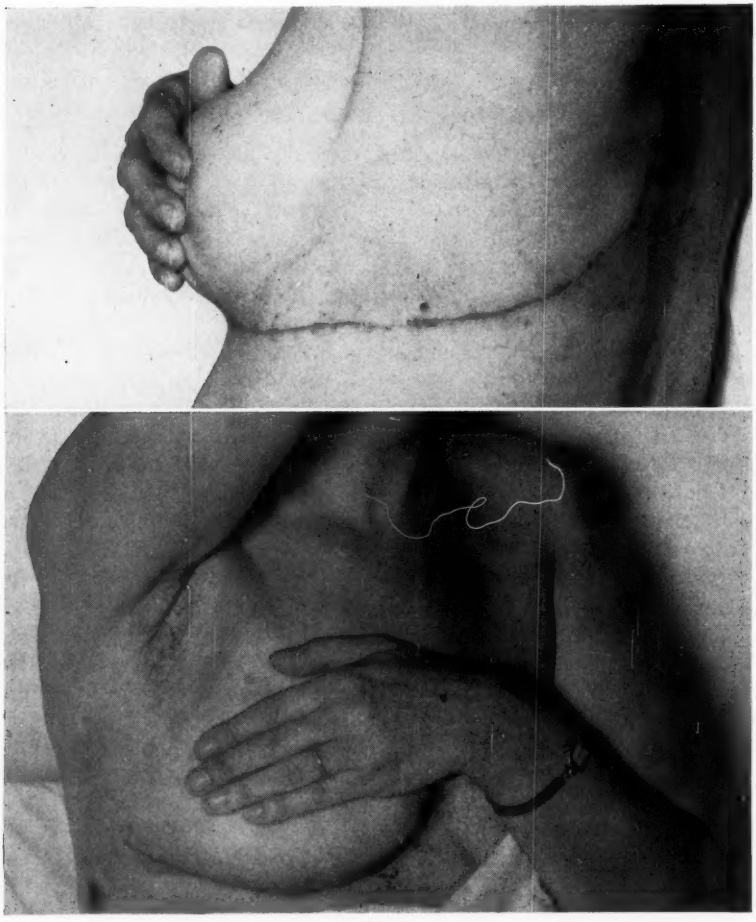
An apparently minor advantage of the anterolateral incision is related to its placement (in the female) largely within or slightly below the inframammary fold. This localization renders the incisional scar scarcely visible in the nude, and completely concealed when the patient wears the usual type of breast support or a modern two-piece bathing suit (Fig. 13, *A* and *B*).

In 25 consecutive female patients operated on through a right-sided incision, the length of the incision was found to average 27 cm. A comparison was made with a similar group of patients in whom the approach to the mitral valve had been made through a left posterolateral wound. The average length of the scars measured 47 cm.

Inasmuch as 71.5 per cent of the patients in whom we have performed commissurotomy for isolated mitral stenosis are females, and since 75.7 per cent of these were below the age of 45, it may not be amiss to consider

seriously the cosmetic aspects of these operations. An incalculable amount of psychologic distress may be imposed upon a sensitive patient by the creation of an ugly or deforming scar.

A.



B.

Fig. 13.—*A*, Long (average 47 cm.) posterolateral incisional scar resulting from customary left-sided operation for mitral stenosis.
B, Cosmetically superior and much shorter (27 cm.) anterolateral incisional scar produced during right-sided approach to the mitral valve. The transaortic approach for aortic stenosis and the natural approach to the tricuspid readily may be made through this same incision.
Courtesy New York J. Med., vol. 56, March, 1956.

OPERATIVE TECHNIQUE FOR MITRAL COMMISSUROTOMY PERFORMED FROM THE RIGHT SIDE

The patient is positioned on his back in the supine position. A small pillow or folded sheet may be placed under the right shoulder to tilt the patient slightly toward the left, and the right arm is abducted 90 degrees.

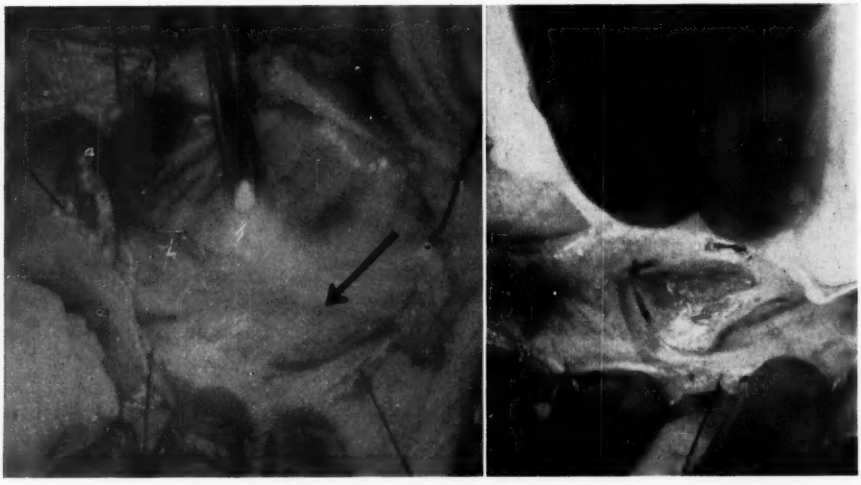
THE THORACIC ENTRANCE.—In the female, a smoothly curved incision is made 1 cm. below the right inframammary fold from the sternal border at the level of the fourth costal cartilage to within 4 cm. of the apex of the axilla. In the male, the incision is made somewhat less curved and passes just below the nipple. The fibers of the pectoralis major and minor are split and divided as necessary to expose the fourth intercostal space. The long thoracic nerve running longitudinally close to the midaxillary line is preserved. The intercostal muscles are incised in the middle of the interspace, and the right pleural cavity is entered. The fourth costal cartilage may be divided at its sternal junction to afford better exposure of the intrathoracic contents. The internal mammary artery and veins are mobilized separately. At this level, it is common to find one vein medial and one vein lateral to the artery. Each vessel is doubly clamped, divided between the clamps, and the cut ends are ligated individually. So treated, few of these vessels will bleed at a later time. The fourth and fifth ribs are covered with large, moist gauze pads and are spread apart to a limited extent by a Finochietto or other type of self-retaining retractor. To allow additional separation of the ribs, the intercostal muscles are divided farther posteriorly along with the pleura. Over a period of twenty minutes, the retractor spread is increased by small increments, thus avoiding rib fracture.

THE CARDIAC APPROACH.—The lung is compressed posteriorly with a large gauze pad, exposing the pericardium. This is incised in the pattern of the capital letter "L" lying on its side. One limb of the pericardial incision runs parallel to the right phrenic nerve and 1 cm. anterior to it, extending from the arch of the aorta superiorly to the lower margin of the right inferior pulmonary vein. The other limb is directed forward at 90 degrees from this latter point across the right atrium until it passes just under the sternal border at the anterior extremity of the wound. Care is taken to ligate all bleeding points along the pericardial edges. Traction sutures are placed in the anterior and posterior lips of the pericardium. The phrenic nerve is avoided assiduously since inadvertently produced diaphragmatic paralysis will result in postoperative persistence of the usually presenting symptom of shortness of breath.

A distinct groove or sulcus can be made out upon close inspection of the surface of the right side of the heart (Fig. 14, *A*). It runs in a cephalocaudad direction, starting just behind the superior vena cava and separating the right atrium, which lies anteriorly, from the left atrium along a line proximal to the junction of the right pulmonary veins. This interatrial sulcus is covered with epicardium and is filled in with adipose tissue so that it is best visualized during atrial systole. This is the same sulcus which is dissected in the Sondergaard technique for closure of interatrial septal defects.[43]

The interatrial boundary may be demonstrated more definitely by making a limited (1.5 to 2 cm.) longitudinal incision in the epicardium over the middle of this lightly colored fat pad, and by continuing this dissection bluntly to a depth of 0.5 cm. (Fig. 14, *B*).

THE ENTRANCE INTO THE LEFT ATRIUM.—A purse-string suture of heavy braided nylon (No. 2) swedged upon a curved needle is placed widely (at a distance of .5 to 1 cm.) about the dissected area. Care is taken that the internal loops are made less than 8 mm. in length, and that the needle point actually penetrates into the lumen of the underlying atrium with each pass. Failure to include the entire thickness of the atrial wall within the suture line will render the attachment of this hemostatic purse string insecure. It is essential also definitely to traverse the underlying interatrial septum with the purse-string suture at the upper and lower ends of the interatrial groove in order to limit any tendency toward "spontaneous" separation of the two halves of the laminated interatrial septum. Reinforcement of this restraining



A. *B.*

Fig. 14.—*A*, Photograph of the right side of the heart at operation, showing location of interatrial groove (arrow).

B, Photograph showing the limited dissection of the interatrial groove and placement of the hemostatic purse-string suture. (Courtesy of A.M.A. Book of Exhibits, 1955.)

effect is accomplished by placing a No. 3-0 cotton stay suture deeply into and across the interatrial groove and its contents just beyond the purse-string suture at each end of the groove. The ends of these cotton sutures are left long after tying and the needles are permitted to remain attached so that they may be used later to oversew the dissected portion of the groove (Fig. 15, *A*).

The long ends of the nylon purse string now are incorporated within a Rumel-Belmont tourniquet in order to apply flexible, dependable, hemostatic tension to the suture line. A heavy braided nylon suture with a swedged-on curved needle is placed in mattress fashion across the area of dissection in such a way that the lips of the wound to be made in the left atrial wall must become everted when it is tied down. The incision will be made within the confines of this mattress suture, care being taken not to interrupt it. By this means no raw wound will be left finally within the left atrial chamber. The dissection of the

groove may be extended and deepened somewhat to admit the tip of the operator's *left* index finger from which the glove finger has been cut away. The bare finger is moistened with a dilute heparin (1/1000) solution and is inserted, palmar surface uppermost, into the apex of the dissection. The end of a broad-bladed knife (Bard-Parker No. 22) is laid flat upon the palmar aspect of the distal phalanx of the left index finger. Fig. 15, *B* shows a special guard devised for the blade by Dr. Richard A. Gilman. This instrument serves to minimize any danger of dividing the purse string and reduces the possibility of penetrating the right atrial chamber inadvertently. This accident occurred three times in our early experience. In each instance, the opening was closed securely with interrupted nylon sutures by a technique reminiscent of the atrio-septo-pexy procedure for interatrial septal defect.[40]

Controlled and guided by the finger, the blade is advanced to pierce the posterior half of the partially separated interatrial septum. It is important to limit the extent to which the knife penetrates within the lumen of the left atrium. Otherwise, there is some chance that it may puncture the posterior (vertebral) wall of this chamber (Fig. 15, *C*, and *D*) and cause severe hemorrhage, the site of which cannot be exposed for suturing except by promptly transecting the sternum, opening the left pleural cavity, and dislocating the heart upward and out of the pericardial sac. The Gilman guard, by limiting the length of blade tip which may emerge from its cover, would seem to obviate this risk.

The entrance of the knife blade into the left atrial chamber is heralded by a small gush of bright red blood. The knife is withdrawn immediately and the finger tip is inserted into the opening to tamponade it. As the finger tip is advanced cautiously, perhaps with a gentle twisting motion, the stab wound in the left hemiseptum gradually becomes dilated to accommodate it (Fig.

TABLE IX. HAZARDS OF RIGHT-SIDED ENTRANCE INTO LEFT ATRIUM AND APPROPRIATE PREVENTIVE AND CORRECTIVE MEASURES

HAZARDS	SAFEGUARDS AND MANAGEMENT
1. Hypotension from pressure on right side of heart	1. Close touch with anesthesiologist; avoidance of prolonged retraction against right atrium; use of vasopressive agents
2. Tearing out of purse string (possible massive hemorrhage)	2. Care to see that purse-string loops penetrate full thickness of atrial wall; approximation of lips of cardiac wound with Nelson tissue forceps if this accident should occur
3. Accidental cutting of purse string (possible massive hemorrhage)	3. Use of Gilman guard (see Fig. 15, *B*) on entering left atrium; approximation of lips of cardiac wound with Nelson tissue forceps
4. Laceration into right as well as left atrium; production of septal defect	4. Care to cut into only *posterior* half of dissected interatrial septum; repair of septal opening by the technique of atrio-septo-pexy
5. Stab wound penetration of posterior wall of left atrium	5. Care not to insert knife blade too deeply (Gilman guard); prompt opening of left pleural space and left side of pericardium to elevate cardiac apex and expose wound

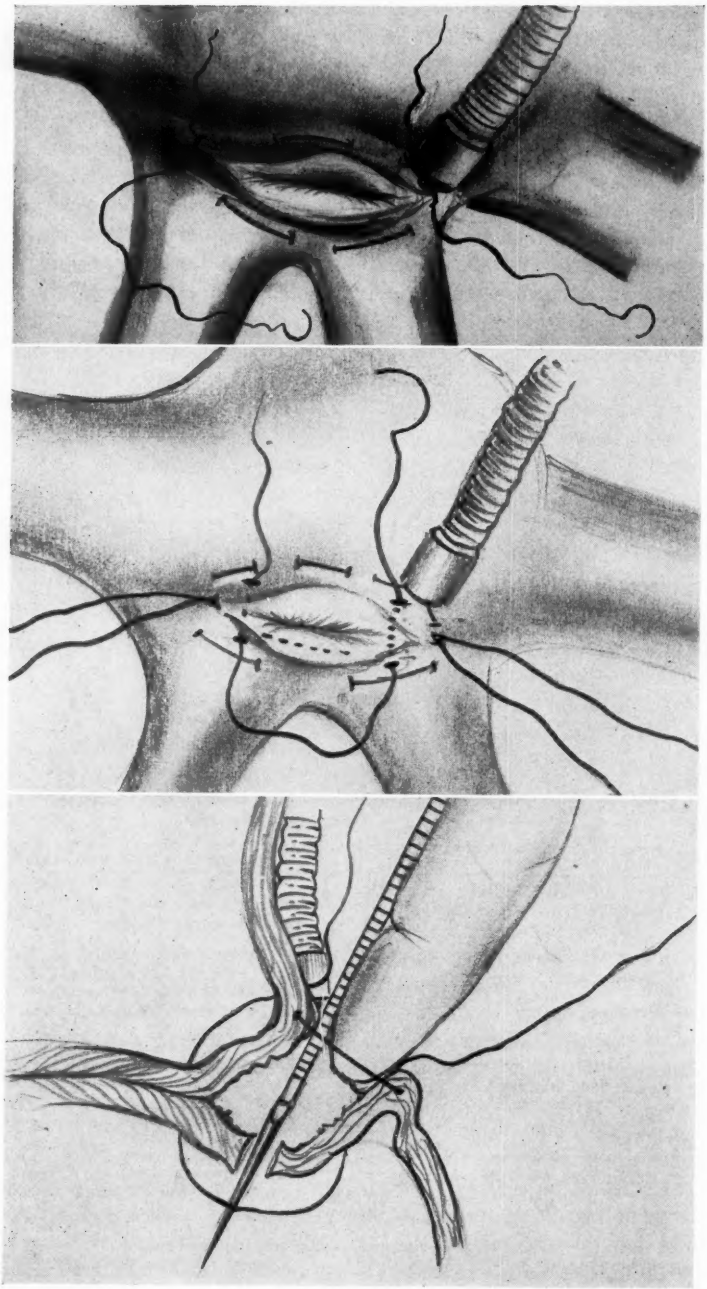
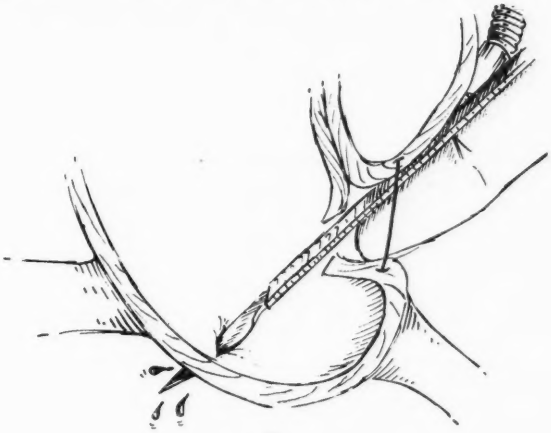


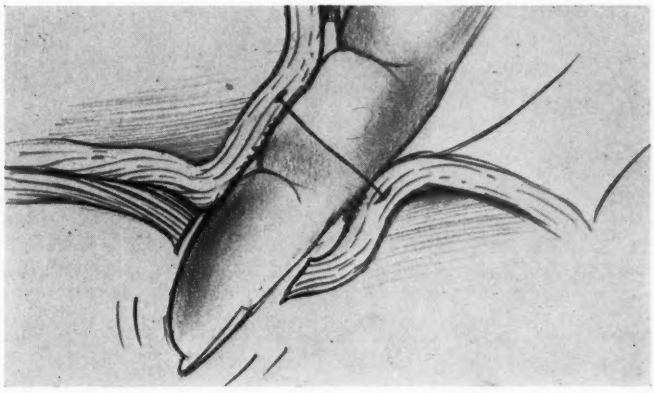
Fig. 15.—*A*, *B*, and *C*. (*For legend see opposite page.*)

15, *E*). The technical hazards associated with the right-sided entrance into the left atrium are listed in Table IX. The treatment as well as the prevention of these errors is suggested.

EXPLORATION OF THE LEFT ATRIUM AND THE MITRAL VALVE.—The valve orifice when so approached lies somewhat farther from the site of cardiac entrance than when explored from the more customary left side. This may necessitate the insertion of the whole length of the finger into the atrial



D.



E.

Fig. 15.—*A*, Illustration showing placement of restraining stay sutures through the partially split atrial septum at either extremity of the dissection.

B, Heavy braided nylon mattress suture placed across dissected region in such a way that tying down will evert lips of left atrial incision (to be made).

C, Penetration of the lower (left) half of the dissected septum with knife blade. This incision is kept within the confines of the mattress suture.

D, Possible perforation of the posterior atrial wall by a simple knife blade if it is inserted too far.

E, Entrance of ungloved tip of left index finger through slitlike puncture opening made in left half of dissected septum. As the finger is advanced in a gentle twisting fashion, the septal incision becomes lengthened somewhat to accommodate it. Usually, tension upon the purse string is not required for hemostasis at this time. (Courtesy New York J. Med., March 15, 1956.)

chamber. During the subsequent maneuvers, care must be taken not to compress the right atrium and venae cavae so firmly as to shut off the flow into the right side of the heart. Even lesser degrees of pressure, if maintained, may bring about severe hypotension, perhaps by a reflex mechanism. When the valve orifice is reached, it is examined digitally. The possible existence of insufficiency is sought for, and if a regurgitant jet is noted, an attempt is made to evaluate its relative size (1 to 4 plus) and to localize it with respect to the position of the extremities of the valve slit. The circumorificial portion of the valve is palpated in an attempt to determine the relative flexibility or rigidity. The presence of any calcification and its amount, localization, and character are noted. In those cases in which the calcific deposition takes the form of loosely attached sandlike particles or even of stalagmites (Fig. 16),

Fig. 16.—Photograph of mitral valve showing loosely attached calcific sand which readily may become detached during the necessary manipulations of the valve.

special care must be taken not to cause encrustations from breaking off during the required valvular manipulations. The incidence of calcification of the mitral valve as determined by digital examination is noted in Table X.

TABLE X. INCIDENCE OF CALCIFICATION OF THE MITRAL VALVE IN 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE COMPARED WITH THE FIRST 1,000 CASES FROM THE LEFT

SIDE	NUMBER OF CASES	NUMBER CALCIFIED	PER CENT
Right	200	85	42.5
Left	1,000	370	37.0

The finger sweeps generally around the lining of the cardiac chamber and explores especially the posterior atrial wall and the region of the left atrial

appendage in a search for intra-atrial thrombotic masses. If such a clot is found, thereafter it is avoided assiduously in order not to dislodge a fragment.

The finger tip now is inserted deeply into the diminutive valve orifice, by the same movement both measuring the size of the opening and, in favorable cases, bringing about the beginning of commissural separation (splitting). As it passes below the apex of the valve cone, the finger tip attempts to palpate the supporting chordae and papillary muscles to determine their location and condition. In no case, should the finger be permitted to block the valve passageway for a longer time than that occupied by three heartbeats. It must be withdrawn completely into the atrial chamber within this period.

After permitting a few seconds for cardiac recovery, the finger reexamines the valve orifice to determine if any effective enlargement has been produced, if any new regurgitation has been created, and to recognize at which commissure the splitting may have started. Should a beginning split be found, in the absence of an increased amount of regurgitation, disruptive digital pressure then is applied directly at the apex of this area of partial valve divulsion.

Digital Commissurotomy ("Finger Fracture").—Since effective external counterpressure over the commissures seldom is feasible when using this right-sided approach to the mitral valve, a different digital technique is employed from that which is customary with the left-sided approach. The apex of the intracardiac finger is placed upon the atrial aspect of the valve at a point just peripheral to one of the extremities of the valve slit. Gradually increasing pressure is applied to the valve margin, being directed toward the apex of the heart. Soon, the bare finger tip will slip over the apex of the valve commissure through the orifice and into the left ventricular chamber. This maneuver momentarily applies considerable but controlled divulsive force to this extremity of the valve without incurring significant risk of injuring the wall of the heart (Fig. 17, *A* and *B*). Basically, it utilizes the principle of the paper knife.

In favorable cases, the fused leaflets will separate a few millimeters with each such stroke, and this may be repeated again and again until the operator is convinced that as complete a separation of this commissure has been brought about as can be accomplished by blunt pressure. When the lengthening of one extremity of the valve slit has been extended fully to the annular ring, which is readily palpable as a circular cordlike structure completely encircling the basal portion of the valve substance, the surgeon's attention must be directed toward the opposite one (Fig. 17, *C*).

Frequently, at one or both commissures, only a limited or no separation may be achieved even by persistent repetition of this maneuver. Generally, this will lead to the conclusion that an instrumental attack upon the valve is indicated.

Creation of Mitral Regurgitation.—In a certain percentage of patients, at some stage in the process of valve splitting, a new regurgitant jet will be detected which, when first noted, will be found to be localized to the apex of the dissection. This jet will be small and of high velocity. However, any

further attempt to achieve additional separation of the valvular tissue at this commissure is almost certain to be attended by a disproportionate increment of increase in the amount of incompetence. Then the regurgitant stream becomes less "jet-like" and more diffuse, involving a greater portion of the valve aperture. It is felt, therefore, that the first recognition of a digitally detectable jet of created regurgitation, occurring at the valve extremity which is being opened, constitutes a contraindication to any further manipulation of this portion of the valve. Its creation probably has been due either to a relatively small deviation of the commissural cleavage from the true

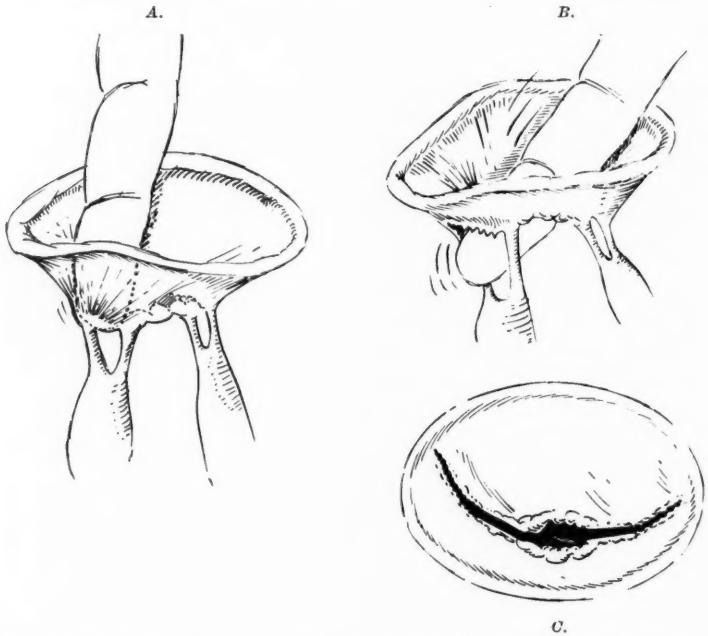


Fig. 17.—Application of divulsive pressure to one of the valve commissures by the right-sided method.

A, Pressure is applied by the finger tip against the fused tissue just beyond the actual valve extremity, being directed toward the ventricle.

B, As the pressure is increased, the finger tip slips toward the apex of the valve slit and through its orifice, tending powerfully to separate or disrupt the valve tissue along the line of fusion (paper-knife effect).

C, Valve separation extended to annular ring. The lines of splitting begin horizontally, but then turn upward at either valve extremity.

anatomic line of rheumatic cusp fusion, or to an extension of the splitting of the valve components beyond the limits of adequate chordal support. However, the appearance of a minor degree of regurgitation at one extremity of the valve should not deter the operator from attempting to separate the opposite commissure. On the other hand, should a major or even a moderate amount of incompetence be created during the surgical mobilization of one commissure, no attempt should be made to open the other one. Such an

effort might well increase the over-all leak to lethal proportions. Furthermore, full separation of the valve tissue will tend to inhibit or to delay the subsequent development of valvular restenosis which here represents our best hope for "spontaneous" correction of the regurgitation.

OBJECTIVE: BICOMMISSURAL MOBILIZATION.—When both commissures have been separated to the valve ring (annulus fibrosus), the opening is considered anatomically to be as large as can be produced in the given patient.

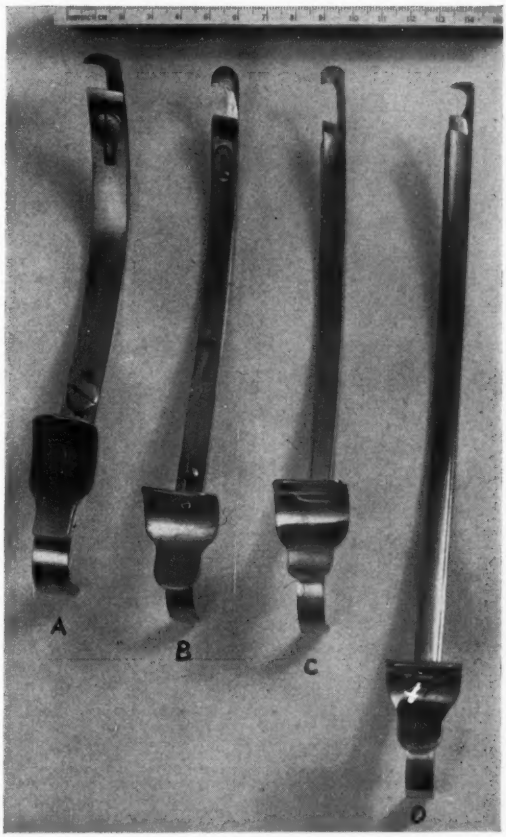


Fig. 18.—Progressive modifications of the guillotine knives used in the performance of mitral commissurotomy.

A, Original primitive broad-bladed guillotine knife designed in 1949.

B, Reduction in size and refinement by O'Neill in 1950.

C, Streamlining of ensheathed guillotine knife using tongue-in-groove principle, by Bailey and Glover in 1951.

D, Lengthening of instrument for use by the right-sided approach and incorporation of the sharp underjaw in conformity with the papillotome principle of Nichols in 1955.

Usually, in the average-sized adult patient, this will be appreciated and designated as a 2 finger opening or larger (estimated 4 to 6.0 sq. cm.). However, with fibrotic contraction or dilatation of the annulus there is corresponding variation in the actual size of the opening which may be achieved.

Unfortunately, extreme fibrosis or calcification of the leaflets may render their motion sluggish, so that the restored functional adequacy of the valve may not be proportional to the anatomic extent of leaflet separation.

When it is impossible to mobilize one or both extremities of the valve fully by blunt digital pressure, instrumental division of the fused commissural substance, or at least of the dense marginal tissue, is necessary. While valvulotomes of various types might well be effective, we nearly always have used somewhat elongated versions of our original right and left guillotine

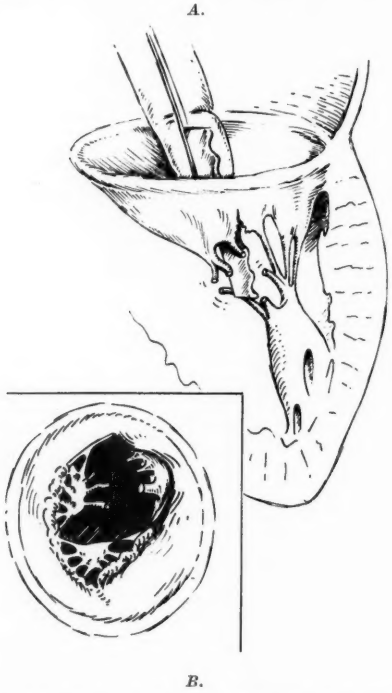


Fig. 19.—*A*, Danger of inadvertent division of the chordae supporting the septal leaflet when the "bread knife" is used to divide a valve commissure. This is an inherent risk associated with the use of any valvulotome so designed that it must be extended deeply below the valve margin, especially when a sawing type of motion is required. This danger is greatest when the papillary muscle has become adherent laterally to the ventricular wall so that the chordae to the septal leaflet run obliquely across (below) the valve orifice. *B*, Insert showing flail-leaflet so produced.

knives. These have been modified as suggested by Nichols[12] so that they may be used also to divide an extensive cross-fusion of the subvalvular supporting tissue (Fig. 18, *A, B, C, D*).* They are inserted individually, in the closed state, into the left atrial chamber by passing them along the palmar surface of the bare left index finger as it extends into the cardiac chamber, while the anterior lip of the dissected interatrial groove is held up by tissue forceps to facilitate the passage. These instruments are so streamlined that they fit the finger smoothly without incurring a significant blood loss.

*Obtainable from George P. Pilling & Son Co., Philadelphia, Pa.

Harken and Black[20] have "violently condemned" the use of the guillotine valvulotome as "indelicate and clumsy," and have recommended instead their "bread knife." While our experience with the latter instrument admittedly is limited, there is unquestionably a serious risk of dividing the supporting chordae to one of the leaflets when the end of such an instrument is passed well below the valve level, as is necessary with any valvulotome which is used in a sawing manner (Fig. 19, *A* and *B*). On the other hand, the tapering probe-like underjaw of the modern guillotine facilitates its close application to the leaflet substance without danger to the chordae (Fig. 20). However, the

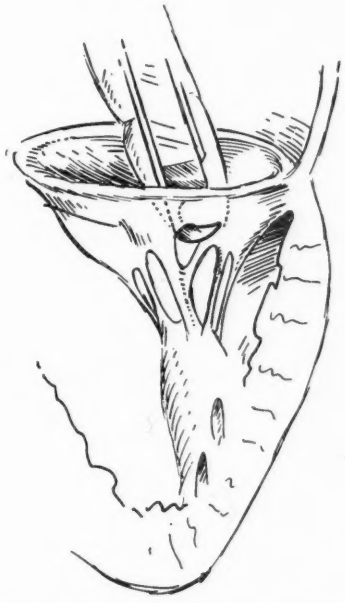


Fig. 20.—In contrast, the very narrow pointed underblade of the modern guillotine knife permits its accurate application immediately against the undersurface of the commissural tissue even when there is shortening of the "yoke" of chordopapillary suspension or oblique chordal suspension.

question is not whether a surgical instrument has been adapted from the French Revolution or merely borrowed from the kitchen, our point is that many different types of valvulotome may be found efficient and safe in the hands of those who have become skilled in their use.

Technique of Instrumental Commissurotomy When Using the Guillotine Knives.—The guillotine may be passed through the valve opening and brought against the apex of the appropriate commissure in the closed state. If this technique is used, it is then advanced sufficiently to extend slightly below the ventricular aspect of the valve substance, and is pressed against the commissure in the line of leaflet fusion. The jaws of the instrument are opened by retracting the sliding blade, thereby permitting the lower blade

to slide beneath the fused commissural tissue. Digital palpation of the undersurface of the valve now establishes that the underjaw of the instrument has been placed accurately in line with and between the two parallel rows of supporting chordae and above any area of possible chordal fusion (Fig. 21, *A*, *B*, *C*).

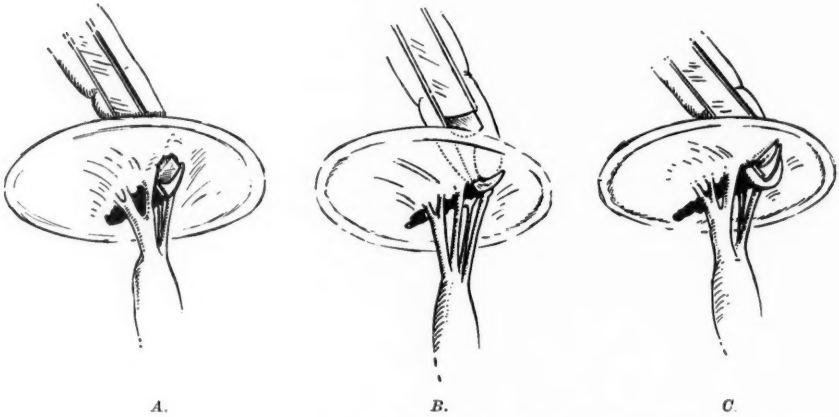


Fig. 21.—Application of the guillotine knife to a fused valve commissure by the closed method.

A, The appropriate right or left instrument is inserted along the palmar surface of the intracardiac finger and is advanced in the closed state down to and through the valve orifice. It is pressed firmly against the apex of the commissure and in line with it.

B, As the sliding blade is retracted, the pointed underjaw slides under the commissure. The finger tip determines that it lies accurately between the two parallel rows of supporting chordae.

C, The sliding blade is driven home by the thumb of the right hand, shearing through 4 mm. of scarred marginal valve tissue.

Fig. 22.—Open technique of engagement of the guillotine knife against the valve commissure. While the instrument is introduced into the heart in the closed state, the jaws are separated before introduction through the valve orifice. By slight angulation, the pointed underblade of the opened instrument may be inserted readily through even the most diminutive orifice and engaged under the fibrosed commissure. Again care is taken that it comes to lie in line with and between the two parallel rows of supporting chordae.

While the authors frequently have used this method of application of the guillotine, they usually have preferred, after introducing the closed instrument into the left atrial lumen, to open its jaws for a distance of approximately 1 cm. before passing the beaked underjaw through the valve orifice and beneath the commissure (Fig. 22). Again, it is stressed that extreme care must be taken to ensure that it has been placed between the two parallel

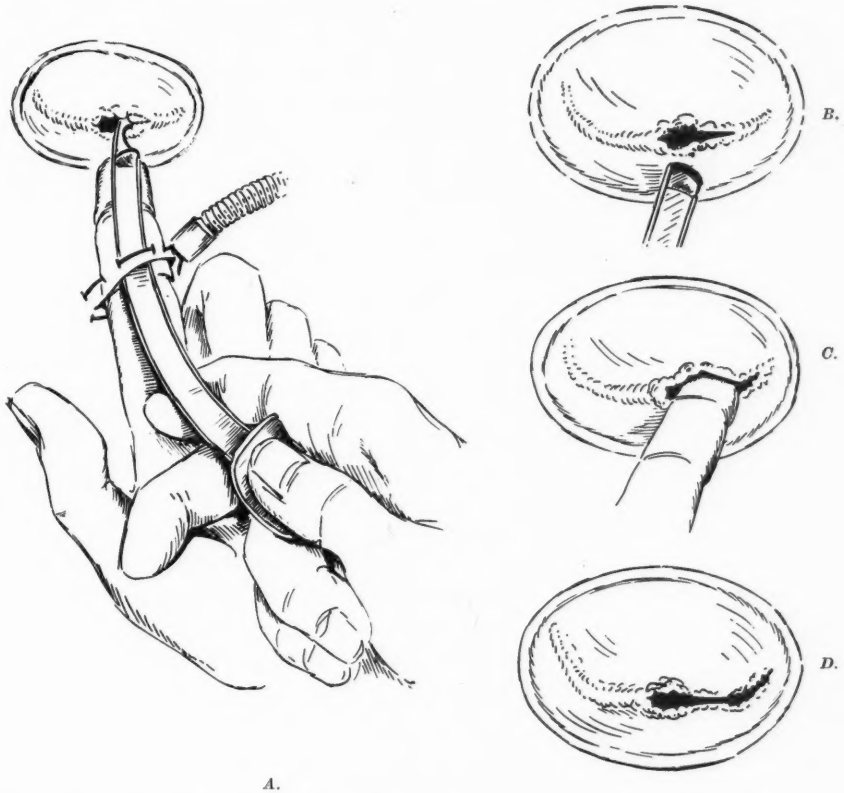


Fig. 23.—*A*, The proper line of direction of instrumental division of the valve will at first lie nearly in the horizontal plane, especially at the posteromedial commissure.

B, Closure of the instrument by advancement of the sliding blade shears through 4 mm. of scarred tissue. The closed head of the valvulotome then is withdrawn from the valve passage completely into the atrial chamber.

C, An attempt at extension of the separation by digital pressure now may prove effective since the continuity of the heavily scarred marginal tissue has been interrupted.

D, If this occurs, the line of subsequent splitting usually will curve upward before reaching the annulus fibrosus.

columns of supporting chordae and in line with them. It must lie above any cross-fused chordae. The plane of the instrument's first cut will be nearly horizontal in the majority of patients.

The operator's right thumb forces the sliding blade home, the index and middle fingers of that hand providing counterpressure against the under-

surface of the curved portion of the handle of the fixed blade (Fig. 23, *A*). The upper blade of the instrument crosses the lower, shearing through 4 mm. of scarred or calcified tissue. With the jaws closed, the lower end of the knife is withdrawn into the atrium and the heart is allowed time to recover from any disturbance incident to the manipulation. We frequently prefer to remove the instrument completely from the heart at this time. However, this will necessitate reinsertion if further cutting should be required. As soon as the anesthetist reports that the level of the blood pressure and the strength of the pulse have been maintained at (or restored to) reasonable levels, the finger tip is advanced to examine the valve with respect to the extent of commissural division and especially to detect any insufficiency which may have been created. Should a new regurgitant jet be found, it nearly always will be minimal, but its very existence should suffice to contraindicate any further manipulation or instrumentation of this portion of the valve mechanism.

Usually, no regurgitation will have been produced, and a further effort at digital splitting of the valve may prove effective now that the continuity of the tough margin of the value orifice has been interrupted (Fig. 23, *B, C*). If a new attempt at digital splitting fails, further instrumentation is indicated until the valve opening extends to the annulus fibrosus, or until the first sign of beginning insufficiency is noted. Should digital pressure become effective at any time (tested by tentative pressure after each episode of instrumentation), it promptly should be substituted for the use of the valvulotome. The line of valve splitting will then normally curve upward (Fig. 23, *D*).

THE POSTEROMEDIAL COMMISSURE.—Not only will the operator find that the posteromedial commissure may be opened readily by the right-sided approach, but it also will be evident that it more appropriately might be designated as the "right" commissure since at this extremity the diminutive valve slit is directed nearly horizontally (then slightly upward) and toward the right. Frequently, the exact line of inflammatory valve fusion will be indicated by a palpable linear elevation or depression upon the atrial surface of the valve which runs from the commissures toward the annulus fibrosus (Fig. 24, *A*). An even more reliable and constant guide to the line of an obliterated commissure is provided by the two parallel rows of chordae which individually support this extremity of each of the respective leaflets. In those relatively few valves in which the subvalvular supports have become agglomerated into massive pillars of fibromuscular tissue (Fig. 24, *B, C, D*) and where the pathologic abnormalities of the atrial surface of the valve obliterate the usual landmarks, experience alone can guide the surgeon in selecting the proper line of division. It is well to remember that the proper line for beginning separation of the right commissure runs essentially in the horizontal plane when the patient lies in the supine position (as stated previously).

THE ANTEROLATERAL COMMISSURE.—It is slightly more difficult to place the anterolateral commissural separation anatomically since the line of fusion (leaflet closure) usually starts horizontally, but soon curves sharply upward.

This may be indicated by palpation of a linear elevation or depression upon the atrial surface of the valve as mentioned previously. This marks the line of fibrous juncture of the cusps. Although the two columns of supporting chordae can be felt below the valve, their complete distribution to the leaflets may be difficult to make out until the valve mobilization has been completed. It is felt that, as with the right commissure, the first incision of the fused

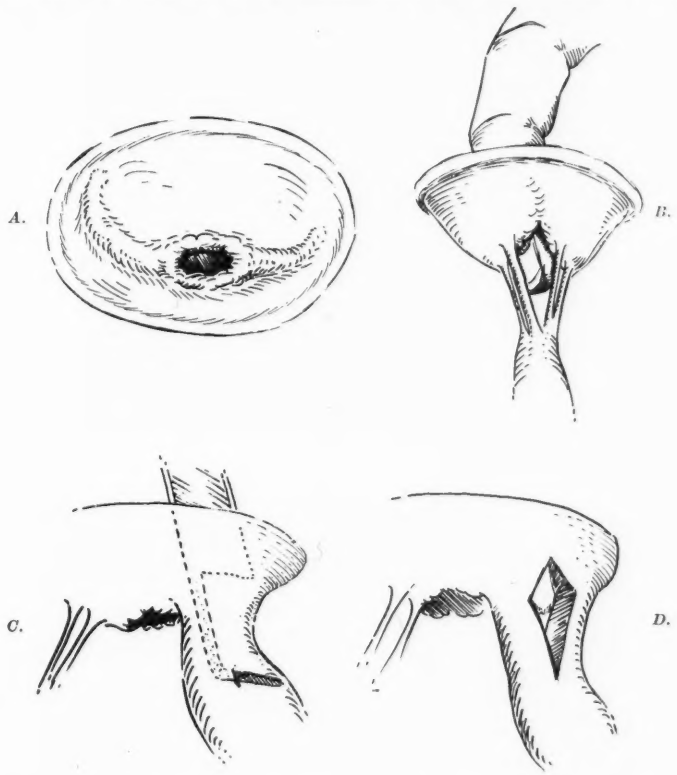


Fig. 24.—*A*, Linear fibrous elevation upon the atrial aspect of the valve which represents the line of leaflet adherence may guide the placement of the valvulotome.

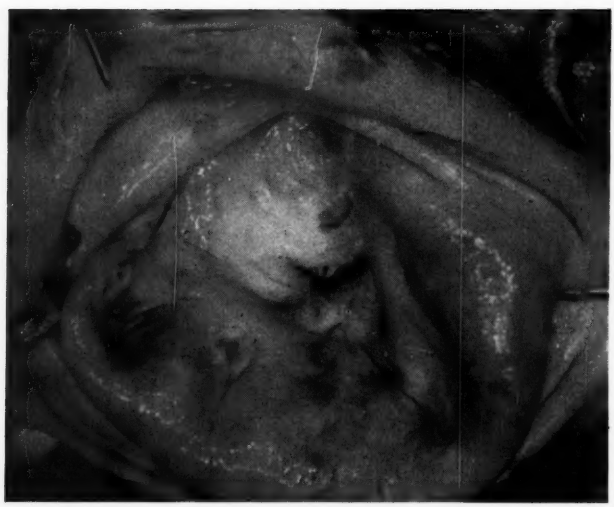
B, Recognition by palpation of the two rows of supporting chordae below the valve similarly may orient the surgeon in his placement of the valvulotome.

C, If there is no linear elevation upon the atrial aspect of the valve, and if there is only a single massive pillarlike fibromuscular support below the valve, the pointed underblade of the instrument simply may be driven deeply into the middle of this pillar in a horizontal direction.

D, Then, closure of the guillotine will start the proper division of both the valve and its support.

anterolateral commissure should be made in a horizontal direction unless a prominent radiating linear elevation or the curve of the valve slit during ventricular systole clearly delineates an immediate upward deflection of the natural line of leaflet separation. A second cut, when necessary, begins at the apex of the first and may be permitted to deviate upward to follow the

A.



B.

Fig. 25.—*A*, Photograph of stenotic mitral valve showing linear depression corresponding to a markedly curved line of valve fusion. (Courtesy Dr. John Storer and New York J. Med., March 15, 1956.)

B, Photograph of stenotic mitral valve showing a lesser arc of curvature which results from a different type of valvular distortion. (Courtesy A.M.A. Book of Exhibits, 1955.)

natural curve of valve approximation. After division of the tough free valve margin anterolaterally, further separation of the tissues often may be accomplished by digital pressure alone. This blunt splitting tends to follow the natural anatomic line of valve fusion (Fig. 25, *A-D*).

Further instrumentation of this extremity of the valve should be continued until digital splitting becomes possible, until a fully adequate valve opening is achieved, until the line of leaflet separation reaches the annulus fibrosus, or until the first sign of created regurgitation at this pole of the

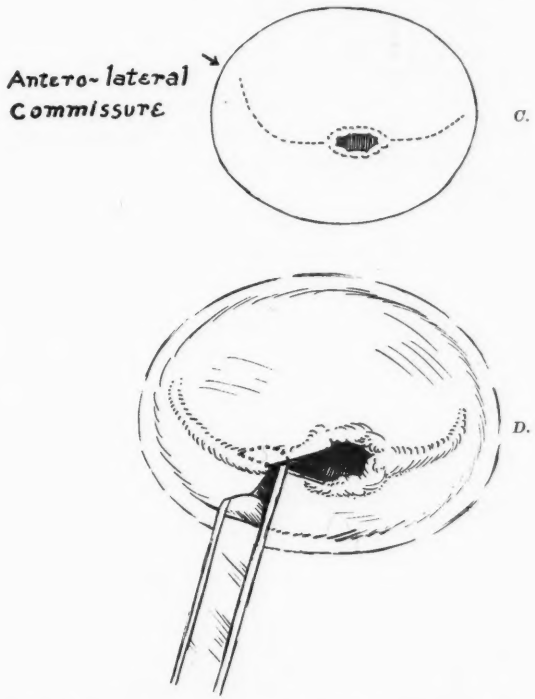


Fig. 25, Cont'd.—*C*, Average line of curvature which the instrumentalist may follow with reasonable confidence in the absence of any more definite guide.

D, Application of open guillotine against the apex of the previous cut for a second one. The first section may have been made horizontally. The subsequent ones should be caused to deviate somewhat in an upward curve.

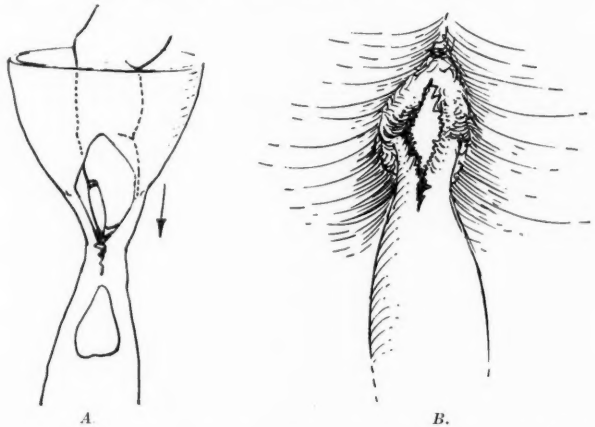
orifice interdicts further efforts. In general, our attempt should be to extend the leaflet mobilization at both extremities fully and naturally to the annulus fibrosus (clearly detectable to palpation), but never beyond this limit.

Table XI indicates the variations in technique of commissural separation which were used in our series. It is understood that in many of the valves the mobilization was begun instrumentally, as indicated, and then was completed by digital pressure (not indicated in the table).

TABLE XI. DIGITAL OR INSTRUMENTAL OPENING OF COMMISSURES IN 200 MITRAL COMMISSUROTOMIES FROM THE RIGHT SIDE

	NUMBER OF CASES
Both commissures opened digitally	28
Anterior commissure opened instrumentally, and posterior digitally	26
Posterior commissure opened instrumentally, and anterior digitally	25
Both commissures opened instrumentally	116
Anterior commissure opened digitally, posterior not separated	2
Anterior commissure opened instrumentally, posterior not separated	1
Posterior commissure opened digitally, anterior not opened	1
Posterior commissure opened instrumentally, anterior not opened	1
Total	200

SUBVALVULAR STENOSIS.—The chordae supporting the respective leaflets were found to be mutually cross-fused in such a way as to reinforce seriously the inflammatory union of the two thickened leaflets in 29 of our first 200 patients with stenotic mitral valves operated on from the right side (Table XII). In some individuals, one or both of the papillary muscles were found to be attached directly to the valve apex (see Fig. 5, *C*). In either instance, after recognition of the condition, the first effort was a cautious but determined effort to separate the leaflets themselves. After the leaflets had been mobilized anatomically an attempt was made by direct downward (toward the apex) pressure of the tip of the bare index finger to separate any remaining fused chordopapillary structures (Fig. 26, *A*).



A *B*.

Fig. 26.—Relief of subvalvular obstruction. *A*, Separation of cross-fused chordae by direct end-on pressure with a finger tip. Often this may be accomplished inadvertently during digital mobilization of the valve commissure, perhaps even without recognition of the condition.

B, A beginning split of a fused papillary mass may be produced during the separation of the commissures.

This program is especially effective against lightly cross-adherent chordae. If the leaflet separation inadvertently (as is usual) has established a beginning division or laceration of the "top" of a complex papillary fusion mass, it may be possible easily to extend this beginning longitudinal "splitting" of the

C. *D. 1* *D. 2*

Fig. 26. Cont'd.—*C*, Such a papillary split may be extended by finger-tip pressure, perhaps to its ventricular attachment. *D, 1* and *2*, In resistant cases, definitive longitudinal division of the papillary mass may be accomplished by boldly piercing it with the sharp-pointed underbeak of the papillotome or modified guillotine knife and then closing the instrument. (Photograph.)

TABLE XII. DISTRIBUTION OF SUBVALVULAR STENOSIS AS RECOGNIZED IN THE FIRST 200 CONSECUTIVE MITRAL COMMISSUROTOMIES PERFORMED FROM THE RIGHT SIDE

		NUMBER OF CASES
Location		
Anterior (chordae only)		9
Posterior (chordae only)		4
Anterior and posterior (chordae only)		6
General fusion and shortening of chordae and papillary muscles (pillarlike masses)		10
	Total	29
Treatment		
Not treated separately—split with leaflet mobilization		12
Treated		17
Split digitally (independently)	11	
Dense papillary adherence		
Cut with guillotine	4	
Cut with papillotome	2	
	Total	29 (14.5%)

E.

Fig. 26, Cont'd.—*E*, Digital splitting of the longitudinally partially divided papillary mass then becomes possible.

papillary muscle mass simply by end-on pressure with the finger tip (Fig. 26, *B*, *C*). In more resistant cases, Nichols'[12] technique of instrumentally starting the separation of the muscle mass may be necessary as it was in 6 of our first 200 patients (Table XII; Fig. 26, *D*, *E*). In some individuals, a subvalvular fusion mass may prove inseparable at one valve extremity, while no subvalvular obstruction may exist at the opposite one, or may readily be overcome. Such resulting eccentric or diagonal entrance passages probably hamper free atrioventricular blood flow to some extent, but in our experience the clinical end result usually has been entirely satisfactory (Fig. 27).

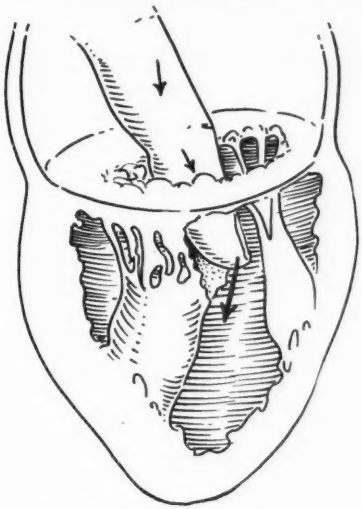


Fig. 27.—Occasionally only an eccentric passageway can be established through a badly distorted valve. However, if the over-all size is adequate, the clinical result usually is good. (Bailey: Surgery of the Heart, courtesy Lea & Febiger, p. 587.)

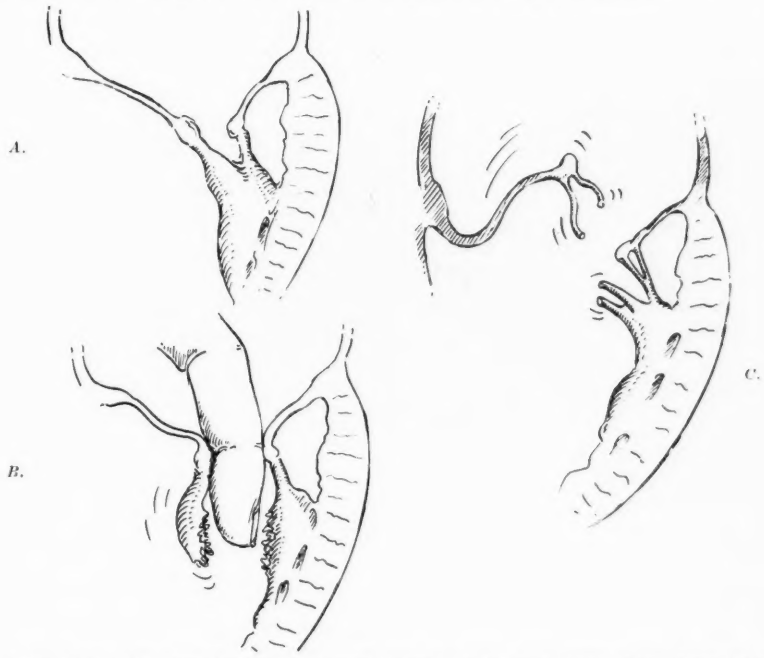


Fig. 28.—*A*, In some instances, extreme lateral fusion may have taken place between a papillary muscle and the ventricular wall. This may be suspected from the course of the chordae which support the septal leaflet.

B, Should splitting of such a papillary muscle prove necessary, great care must be taken to so direct the line of created separation anatomically lest one side of the split muscle may become entirely detached with ensuing loss of valvular support and consequent severe regurgitation.

C, In no instance should any such obliquely or transversely directed chordae be sectioned lest a flail valve be produced.

Normally, both papillary muscles arise from and are attached to the mural (lateral) wall of the left ventricle, fairly well down toward the cardiac apex. In some of these rheumatic patients, a variable amount of lateral adherence between these muscles and the endocardial surface of the lateral wall of the ventricle will have taken place, so that the muscles appear to have a higher (toward the atrium) attachment and to be directed more obliquely with respect to the atrioventricular channel than is normal. This morbid anatomic possibility must be taken into consideration when attempting to split digitally or to divide instrumentally such a muscle mass in its longitudinal axis (Fig. 28, *A*, *B*).

Furthermore, it is essential to realize that when one or both papillary muscles are directed thus obliquely toward the valve orifice, the corresponding chordae which attach to the septal leaflet will be bound to "run across" the valve passageway. Since they support this leaflet, they must not be divided lest severe regurgitation be created (Fig. 28, *C*). In these cases, any long cutting type of instrument (such as the "bread knife") which must be passed deeply below the valve level may be dangerous (see Fig. 19).

Closure of the Dissected Interatrial Groove.—After the definitive valvular surgery has been completed either to the surgeon's complete satisfaction or at the level of the best compromise possible under the existing circumstances, the intracardiac operating finger is withdrawn cautiously as the purse-string suture is tightened progressively. If there is any persistent bleeding, the two lips of the dissected interatrial groove may be approximated temporarily for hemostasis with a pair of Nelson tissue forceps (Fig. 29).

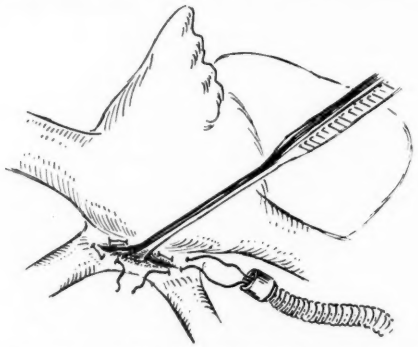


Fig. 29.—Method of control of bleeding between the lips of the dissected interatrial groove. This simple measure may prove lifesaving should the purse-string suture become divided.

This simple measure may prove lifesaving in the event of inadvertent section of the encircling hemostatic purse-string suture. The ends of the previously placed nylon mattress suture now are tied down, bringing about eversion of the lips of the left atrial incision and assuring accurate endocardial apposition within the atrial chamber.

The two lips of the dissected groove are reapproximated with a continuous horizontal everting mattress suture, using one of the No. 3-0 cotton stay sutures with swedged-on curved needles which were placed at each end

of the groove upon the completion of the dissection. After this first suture has been run the length of the cardiac wound, it is tied to the short end of the similar stitch which has been placed at the opposite extremity. The needle-bearing end of this latter suture is used to reinforce the placed mattress layer in an over-and-over running fashion. Its lead end, in turn, is tied to the short end of the first (Fig. 30, *A*, *B*). As the lips of dissection are approximated, the tourniquet tension upon the purse string is released to avoid puckering

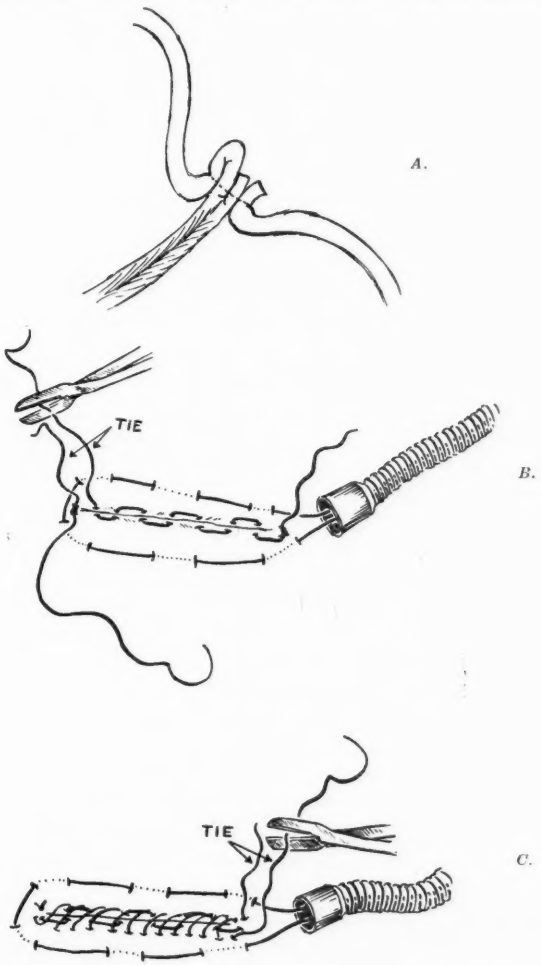


Fig. 30.—Closure of the cardiac wound.

A, Tying down the deep nylon mattress suture everts the lips of the left atrial incision.

B, One of the stay sutures at the lower extremity of the dissection is used to place a continuous mattress suture line which approximates the two lips of the cardiac wound. The leading end of this suture is tied to the short end of the opposite one.

C, The long (needle-bearing) end of this second stay suture is then used to place a row of continuous running stitches just superficial to the mattress layer. It, in turn, is tied to the short end of the first stay suture. The purse string is removed completely, if possible.

of the inner surface of the left atrial wall. We prefer to remove the purse-string suture completely, but should bleeding recur when the purse string is released tentatively, it may be tied or additional sutures may be placed.

TRICUSPID EXPLORATION.—Tricuspid exploration by passage of the left index finger through an incision made in the right auricular appendage is carried out routinely following the mitral commissurotomy. This order of intervention enables the surgeon to be reassured by a second check from another chamber that an atrial septal defect has not been overlooked or created inadvertently during the dissection of the interatrial septum, and permits an immediate repair by the technique of atrio-septo-pexy[41] should one be found.

COMPLETION OF THE OPERATION.—Any necessary tricuspid or aortic surgery for a concomitant lesion having been completed, the pericardium is closed partially with interrupted sutures. This repair must be sufficiently complete to prevent cardiac herniation, but loose enough to permit free drainage of blood and exudate into the pleural cavity. The chest wall is repaired in layers after careful attention has been given to hemostasis. The pleural cavity is drained through a stab wound made in the right seventh intercostal space in the posterior axillary line by a No. 26 Fr. urethral catheter with additional windows cut into it. It is connected to a simple waterseal drainage system.

OPERATIVE MORTALITY

Among the first 130 patients (Table XIII) who were operated on by the right-sided approach essentially for isolated mitral stenosis (with or without associated insignificant mitral insufficiency), there were ten hospital deaths (8 per cent). This was almost identical to that experienced with our first 1,200 patients who were operated on from the left side. The clinical impression of our medical associates has been that an appreciably greater percentage of far-advanced cases has been included in the right-sided group. Eight of these deaths occurred among the first 70 operations while we were relatively inexperienced in the technical aspects of this then new and unusual approach. There have been only two deaths among the last 60 patients. There has been only one late death in this series (Patient S. S.); this was due to massive pulmonary embolization in an asthenic, elderly (58-year-old) patient with extremely advanced renal disease.

TABLE XIII. CAUSES OF OPERATIVE AND LATE DEATH IN 11 MITRAL CASES OPERATED ON FROM THE RIGHT SIDE IN 130 CASES OF PREDOMINANT MITRAL STENOSIS WITHOUT SIGNIFICANT REGURGITATION, TRICUSPID, OR AORTIC DISEASE

HOSPITAL DEATHS	NUMBER OF CASES
Mitral insufficiency created or increased	5
Systemic arterial embolism	
Operative (calcific)	1
Postoperative (thrombotic)	2
Massive hemorrhage	
Technical accident (purse string tore out)	1
Unknown (unstable circulation—hypotension)	1
Total	10 (8%)
Late pulmonary embolization	1

The over-all operative mortality in the entire 200 patients (Table XIV) with all concomitantly performed procedures was 11 per cent (22). One was an additional late death in a patient in whom aortic commissurotomy was performed simultaneously, due to the late development (six months) of subacute bacterial endocarditis.

TABLE XIV. MORTALITY (EARLY AND LATE) IN 200 OPERATIONS FOR ISOLATED AND COMPLICATED MITRAL STENOSIS USING THE RIGHT-SIDED APPROACH

	NUMBER OF CASES	NUMBER OF DEATHS
Complicated Mitral Stenosis		
Aortic commissurotomy	28	4
Aortic annulus constriction for insufficiency	2	1
Tricuspid commissurotomy (175 explored)		
For major stenosis	19	5
For minor commissural fusion	13	-
Tricuspid annulus plication for insufficiency	2	1
Atrio-septo-pexy (for congenital defect)	2	-
Atrio-septo-pexy with surgical transposition of anomalous pulmonary veins	1	-
Right middle lobe resection for arteriovenous fistula	1	-
Mitral commissurotomy in the presence of severe mitral insufficiency (not treated at this same operation)	2	-
Isolated Mitral Stenosis		
Mitral commissurotomy for pure or predominant mitral stenosis and insufficiency, not excluding minor mitral insufficiency or tricuspid insufficiency	130	11
Total	200	22

Three of the 4 patients in whom definitive trivalvular surgery was carried out died. In each, the death was attributed chiefly to the extremely poor condition of the individual.

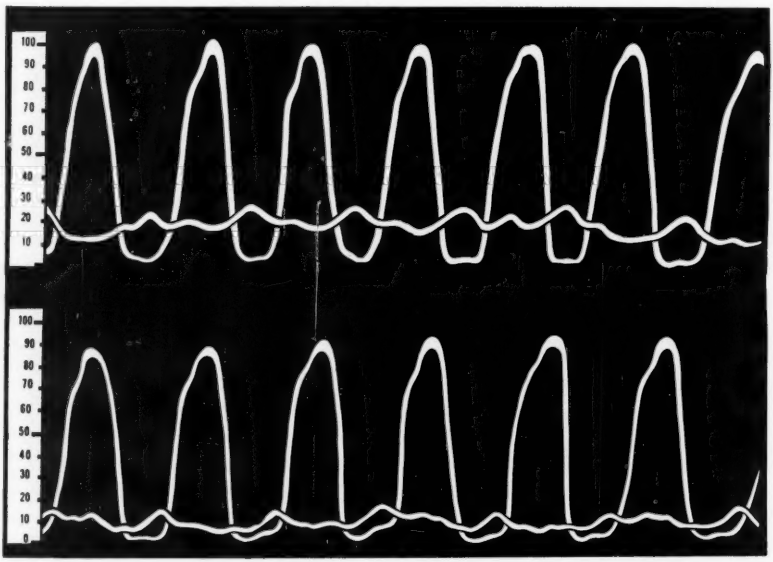
EARLY FOLLOW-UP ON SURVIVING PATIENTS

The early postoperative results in this group of cases have been uniformly excellent, although it must be remembered that the longest period since operation is sixteen months. A personal checkup or a physician's report has been obtained in each patient within the past two months. A preliminary survey of 120 patients surviving operation for mitral stenosis pure or predominant (with insignificant mitral insufficiency) through the right-sided approach revealed only one late death due to massive pulmonary embolism (Patient, S. S.). One other case has been unimproved following surgery (Patient, M. F.). This we feel is related to rupture of chordae tendineae and the production of mitral insufficiency during an attempt to palpate the aortic valve by way of the mitral orifice.

Diaphragmatic paralysis on the right side, apparently due to injury to the phrenic nerve, has handicapped another patient in attaining a complete recovery (Patient, H. S.). Seven patients have experienced one or more episodes of indeterminate malaise, usually appearing four weeks to eight months postoperatively and associated with fever and chest pain, sometimes with detectable pleural effusion. Conservative treatment with acetylsalicylic acid (aspirin) or adrenal corticoid substance (Meticorten) has sufficed in most instances. Thoracentesis has been necessary on occasion.

AUSCULTATORY CHANGES.—Seventy-two of the patients who were operated on for mitral stenosis only were available for personal re-examination by the members of the cardiological staff of our Clinic, three months or more after their surgery. A diastolic murmur at the apex, present in all patients preoperatively, could be heard in only 40 per cent (Table XV). This is in extreme contrast to the general experience following mitral commissurotomy performed from the left side in which the diastolic murmur is abolished in only a minority of instances. Its persistence in 40 per cent of our patients was felt, for the most part, to be related to turbulence of the increased blood flow over rough valve surfaces rather than to any residual obstruction. These impressions were borne out further by comparison of the pre- and postoperative measurement of the diastolic gradient across the mitral valve (Fig. 31, *A, B.*). Left heart catheterization or combined left and right heart catheterization was carried out in over one half of these patients and is being reported elsewhere.[44]

A.



B.

Fig. 31.—Reduction of mitral diastolic pressure gradient as a result of commissurotomy. Left heart catheterization in Patient A. S. (Courtesy of A.M.A. Book of Exhibits, 1955).

A, Note the typical abnormal elevation of the gradient which exists during the period of ventricular filling, indicating mitral stenosis.

B, Marked reduction in the gradient in this patient following adequate mitral commissurotomy.

A less reassuring finding has been the appearance of a systolic murmur in many patients who did not present one prior to surgery. Such a murmur existed in only 25 per cent, preoperatively, but was noted in 57 per cent, three months later (Table XV). There was no correlation between the

TABLE XV. MURMURS AUDIBLE 3 MONTHS POSTOPERATIVELY IN 72 CASES OF PREDOMINANT MITRAL STENOSIS OPERATED ON FROM THE RIGHT SIDE

		NUMBER OF CASES	PER CENT
	Diastolic		
	None	43	60
Present	I - 26 II - 3 III - 0 IV - 0	29	40
	Systolic		
	None	31	43
Present	I - 15 II - 23 III - 3 IV - 0	41	57

Note: The above Roman numerals refer to the grade of intensity of the murmur and not to classification of the cases.

appearance of the murmur and the production of digitally detectable mitral regurgitation at the time of surgery. The murmur was not associated with any compromise in the generally excellent clinical response.

While the postoperative period in many of these patients is very short, a dramatic clinical improvement has been evidenced already in the great majority. One hundred and four of the 111 attending physicians contacted have been most enthusiastic in their reports. The majority of the patients have returned to greatly increased activity and many to full-time work.

SUMMARY

As a result of the rare necessity for the performance of right thoracic exploration in patients with severe mitral stenosis, a simple and satisfactory method has been devised for digital and instrumental entrance into the left atrium from the right side. Increasing familiarity with and improvement in the technique of performing mitral commissurotomy from the right have led to its adoption as our routine operation for most patients with this condition.

Our experience in the first 200 patients so operated upon clearly indicates that the right-sided approach, in contrast to the more usual left-sided one, nearly always permits the accomplishment of a superior type of mobilization of the fused valve structures. In this series, both commissures were divided in 97.5 per cent of the patients. At the same time, it was found distinctly easier to obtain definitive separation of cross-fused subvalvular supporting tissues when present.

The right-sided approach lends itself particularly well to the simultaneous performance of multivalvular surgery in those patients with associated rheumatic disease of the aortic or tricuspid valve. Indeed, the ease of tricuspid exploration is so great that this maneuver has been carried out as a routine at the conclusion of the mitral operation. A surprising number of "silent" but severe stenoses of the tricuspid valve are uncovered thereby, and then may be corrected promptly.

The high frequency of occurrence of thrombosis of the left auricular appendage (29 per cent in 1,200 patients operated on from the left side), and the associated risk of causation of arterial embolization by inadvertent dislodgment of a thrombotic fragment strongly urge the use of some other site of entrance than the appendage into the left atrial lumen for the performance of mitral commissurotomy. The other possible left-sided routes of entry (the pulmonary veins and the atrial wall itself[8, 16]) leave much to be desired both in safety to the patient and in the facility with which the necessary technical procedures may be carried out upon the valve.

The frequency of re-establishment of mitral valvular obstruction, following clinically successful commissurotomy, is related both to the existing pathologic condition of the valve and to the adequacy of the valvular mobilization at the time of the first procedure. The better separation of the valve structures which can be obtained by the right-sided method would seem to commend its initial use in an attempt to obviate the latter etiologic entity. Moreover, once restenosis has become established, a secondary approach to the left atrium through the nearly virginal right thorax and the superior type of valve operation which then becomes feasible would seem to offer the patient the best possible chance for permanent or prolonged relief of the obstruction.

There are many relatively minor advantages associated with the right-sided operation for mitral stenosis which in the aggregate or in the individual case may be of great importance. These include the avoidance of certain left-sided thoracic lesions, the possibility of simultaneously correcting certain right-sided ones, the prevention of severe operative pain and disability which may lead to limitation of shoulder and muscle function, and the superior cosmetic aspects of the right submammary incision.

CONCLUSION

After an experience comprising the performance of mitral commissurotomy for stenosis from the left side in over 1,400 patients, and from the right side in over 200, it is the opinion of the authors that the latter procedure in most cases is, in highly trained hands, a superior one. This applies both to the initial technical accomplishment upon the valve, and also to the operator's versatility of application with respect to many frequently encountered associated involvements and complications. In comparably severe cases, it appears to be an appreciably safer operation than the left-sided one which requires a left lateral position during surgery. It has been applied repeatedly in individuals who were considered too ill for the older type of operation.

The procedure requires a high degree of technical skill for its proper and safe performance and must not be undertaken by the tyro.

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INVOLVEMENT OF THE THORACIC WALL IN BRONCHOGENIC CARCINOMA

Study of 16 Cases in Which Pneumonectomy or Lobectomy and Simultaneous Resection of the Thoracic Wall Were Done*

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PROGRESS in the treatment of patients who have carcinoma of the lung demands careful appraisal of all the factors affecting treatment and prognosis. The literature concerning surgical management of bronchogenic carcinoma with direct extension to the thoracic wall is limited. It is hoped, therefore, that this study might add to the understanding of this problem.

REVIEW OF LITERATURE

Coleman, in 1947, reported 5 cases in which pneumonectomy or lobectomy was accompanied by simultaneous resection of the thoracic wall. Pneumonectomy was done in 4 of these cases and lobectomy in the remaining one. One operative death occurred in his series and the patient who had undergone the lobectomy died 11 months after operation. According to Meade, 2 of Coleman's patients were still living and well 8 and 13 years respectively, after operation. Björk mentioned 2 cases of this type in his study. One of the patients died on the third day after operation and the other survived only 4 months after surgical intervention. Overholt and Schmidt, in 1949, stated that their series included 6 cases of this kind; none of their patients survived for more than 27 months. Gibbon and associates, in 1953, reported a series of 13 cases in which resection of the thoracic wall was performed; none of these patients survived for more than 18 months.

MATERIAL AND METHODS

The first pneumonectomy for bronchogenic carcinoma at the Mayo Clinic was done in May, 1935. The records of pneumonectomies and lobectomies for bronchogenic carcinoma from that date to November, 1954, were searched in order to find the cases in which simultaneous resection of the thoracic wall was done. Sixteen such cases were found; they had been encountered between December, 1947, and October, 1954.

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In all cases, gross specimens were examined and sections were made for histologic study. The sections were stained with hematoxylin and eosin. The cases were classified according to the method presented in 1951 by two of us (McDonald and Clagett) and associates.[3, 8, 12, 13] The malignancy of the tumors was graded histologically after the method of Broders. To determine the extent of carcinomatous invasion, multiple blocks were taken in each instance from the parietal pleura and from the intercostal muscle bundles.

In this study, the term "involvement of the thoracic wall" indicates direct and microscopically verified extension of the tumor to the parietal pleura, with or without invasion of the intercostal muscle bundles and ribs.

Frozen sections of the regional lymph nodes are examined routinely at the clinic by the surgical pathologists. These specimens usually are discarded after this examination. Therefore, the presence or absence of carcinomatous involvement of the regional nodes is recorded in this study as noted in the original report of the surgical pathologist.

The clinical features of each case were reviewed and follow-up data were obtained.

CLINICAL FINDINGS

A summary of some of the data in our 16 patients is presented in the accompanying table (Table I).

Age and Sex.—Thirteen of the tumors occurred in men and three in women. The ages of the patients when first seen at the clinic ranged from 35 to 82 years, but only one of the patients was less than 40 years of age. The average age was 57 years.

Symptoms and Signs.—Pain in the chest, back, shoulder, or axilla was the most prominent symptom. It was the main symptom that forced the patient to see the physician in fourteen instances. Pain was preceded by weakness in two instances and by "cold and cough," hemoptysis and osteoarthropathy each in one instance. The average duration of pain before the establishment of the definitive diagnosis was 4 months, with a range of 2 weeks to 15 months. Only 2 of the 16 patients did not complain of pain; one of these had been feeling "all in" for 4 months and the other had had a cough for 6 weeks.

The findings on general physical examination usually were rather indefinite. Some dullness to percussion and diminution in breath sounds on the affected side of the thorax were present in 6 patients. Tender zones on the thoracic wall were found in 2 patients. Results of physical examination of the thorax were essentially normal in the remaining patients, who made up one half of the entire group. No typical examples of Pancoast's syndrome were seen in this series.

Roentgenographic Findings.—Rather sharply circumscribed shadows usually were found in roentgenograms of the thorax (Fig. 1, *a* and *b*). Erosion of ribs was detected in 4 cases.

TABLE I. RESECTION OF INVOLVED THORACIC WALL IN BRONCHOGENIC CARCINOMA: 16 CASES

CASE	SEX	AGE (YEARS)	MAIN SYMPTOM AND DURATION	OPERATION*	TYPE OF CARCINOMA	INVOLVEMENT OF INTERCOSTAL NERVES AND VESSELS	FOLLOW-UP, TIME POST-OPERATIVE
1	M	50	Pain, 4 mos.	RUL + RTW	Large-cell	+	Died, 14 mos.
2	F	63	Weakness, 4 mos.	LLL + RTW	Adenoca., grade 4	Only parietal pleura involved	Living and well, 6½ yrs.
3	M	47	Cough, 6 wks.	LUL + RTW	Large-cell	+	Died, 2 mos.
4	F	66	Pain, 1 mo.	LP + RTW	Adenoca., grade 3	+	Died, 14 mos.
5	M	63	Pain, 6 mos.	RUL + RTW	Squamous-cell, grade 4	+	Died, 1 yr.
6	M	50	Pain, 2 mos.	LP + RTW	Squamous-cell, grade 4	+	Died, 5 mos.
7	M	65	Pain, 1 mo.	RP + RTW	Large-cell	+	Died, 3 wks.
8	M	65	Pain, 15 mos.	LUL + RTW	Squamous-cell, grade 4	+	Died, 6 mos.
9	M	46	Pain, 2 wks.	RP + RTW	Large-cell	Only parietal pleura involved	Living and well, 2⅛ yrs.
10	M	52	Pain 1 yr.	LP + RTW	Mucous adenoca., grade 4	+	Died, 6 mos.
11	F	82	Pain, 3 wks.	LLL + RTW	Large-cell	+	Died, 1 yr.
12	M	68	Pain, 5 mos.	RLL + RTW	Squamous-cell, grade 4	+	Living and well, 1⅛ yrs.
13	M	43	Pain, 5 wks.	RUL + sup. segm. RLL + RTW	Large-cell	Only parietal pleura involved	Living and well, 13 mos.
14	M	35	Pain, 2½ mos.	RUL + RTW	Adenoca., grade 4	Involvement of brachial plexus	Died, 2 mos.
15	M	60	Pain, 2 mos.	RP + RTW	Squamous-cell, grade 4	+	Living, not well, 8 mos.
16	M	66	Pain, 6 wks.	RP + RTW	Squamous-cell, grade 4	+	Living and well, 5 mos.

*RUL = right upper lobectomy; LUL = left upper lobectomy; RLL = right lower lobectomy; LLL = left lower lobectomy; RTW = resection of thoracic wall; LP = left pneumonectomy; RP = right pneumonectomy.

Bronchoscopic Examination.—Bronchoscopic examination was performed in 7 cases but did not prove to be of help in establishing a definitive diagnosis. Some degree of narrowing of the bronchus of the involved lobe was found in 2 cases.

Examination of Sputum.—Malignant cells were found in 4 of the 11 cases in which cytologic studies of the sputum were done.

OPERATIVE PROCEDURE AND POSTOPERATIVE MORBIDITY AND MORTALITY

A posterolateral incision was made in each instance on the affected side of the thorax. The sixth rib usually was resected and the pleural cavity was entered through the bed of the resected rib. Pneumonectomy was performed in 7 cases and lobectomy in 9. The involved part of the thoracic wall was resected in continuity with the tumor in 14 instances. En bloc resection was not possible in 2 cases; in one (Case 2), the involved portion of the thoracic wall broke loose from the lung, whereas the tumor in the other (Case 14) had invaded the brachial plexus. In the latter case, the tumor was shaved from the plexus as a palliative procedure.

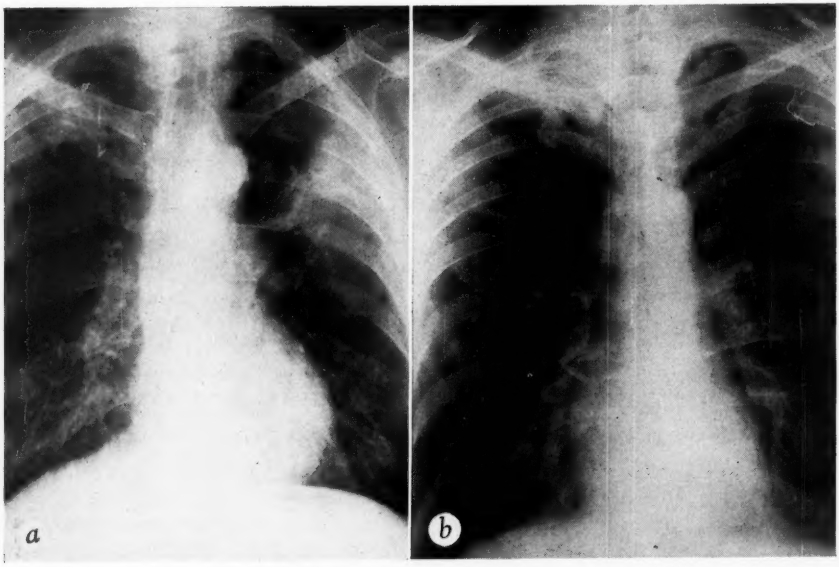

Fig. 1.—*a*, (Case 8), Patient had had pain in the left side of the thorax for 15 months. Note the large peripheral tumor in the left upper lobe.
b, (Case 9), This patient had had pain in the right scapula, shoulder, and arm for 2 weeks. Note the small tumor in the right apex.

The involved ribs and the intercostal muscle bundles were resected in one piece during resection of the thoracic wall. The resection was carried out at least 2 cm. beyond the edge of the macroscopic invasion by the carcinoma in all but 2 cases, in which such resection was impossible because of additional involvement. The brachial plexus was involved in one of these instances (Case 14), and a vertebra was involved in the other (Case 7). The operation in both of these cases was considered by the surgeon to be palliative in nature.

The dead space was filled in 2 instances (Cases 6 and 11) by means of Ivalon sponge. Prolonged postoperative serous drainage developed in one patient (Case 11). This drainage, however, ceased spontaneously. No special measures were used in any other patients to fill the dead spaces and in no instance were any disturbances noted in the healing of the thoracotomy incision.

Most of the patients recovered nicely from the operation. Iliofemoral thrombophlebitis developed during the postoperative period in one patient (Case 12) but he recovered. There were no hospital deaths but one patient (Case 7) died 3 weeks after operation.

Several patients complained of an intercostal neuralgia-like pain on the side of the operation for several months after the surgical procedure. It is impossible to estimate the duration of this pain because of the short length of life after the operation in most instances. One patient (Case 2), who is living and well 6½ years after operation, no longer has intercostal pain; another patient (Case 9) who has been followed for 2⅓ years without evidence of recurrence is nearly completely relieved of the pain.

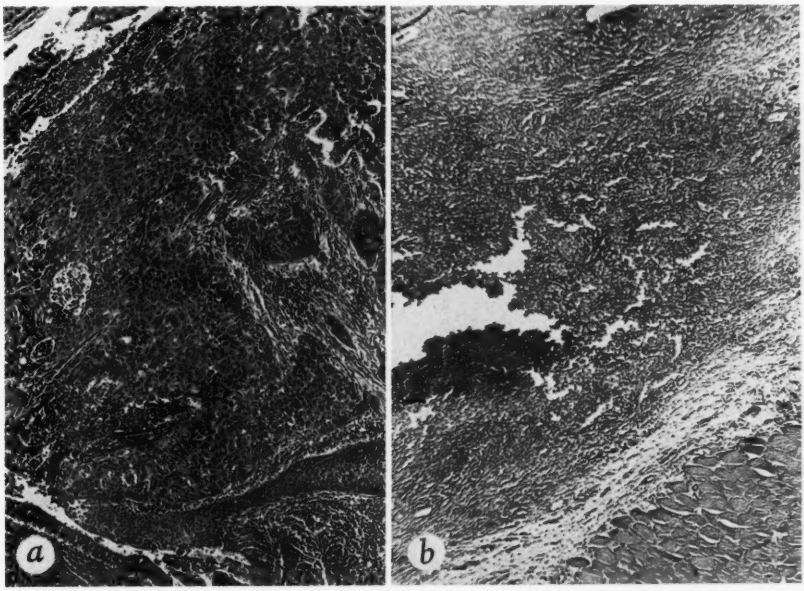

Fig. 2.—*a*, (Case 8), Grade 4 squamous-cell carcinoma (hematoxylin and eosin; ×40). *b*, (Case 9), This tumor was a large-cell undifferentiated carcinoma that had involved only the parietal pleura. Note intercostal muscle in right lower corner and parietal pleura in left upper corner (hematoxylin and eosin; ×30).

RADIATION THERAPY

Three patients (Cases 5, 9, and 13) who did not have any evidence of metastasis received x-ray treatment immediately after their recovery from the surgical procedure. Three other patients (Cases 1, 11, and 14) received radiation therapy for evident metastatic disease. All 3 of these latter patients died of metastasis.

No conclusions can be drawn regarding the efficacy of radiation therapy in the treatment of bronchogenic carcinoma on the basis of this study.

PATHOLOGIC FINDINGS

The tumors in this series were all rather sharply circumscribed peripheral carcinomas. Their diameters ranged from 3 to 8 cm.

In 9 of the 16 cases, the tumor was situated in the right lung; the right upper lobe was affected in 7 of these cases and the right lower lobe in 2. Of the seven tumors that occurred on the left side, four were in the left upper lobe and three in the left lower lobe.

Histologically, all the tumors in this series were anaplastic lesions of a high grade of malignancy. Six of them were grade 4 squamous-cell carcinomas (Fig. 2, *a*), six were large-cell undifferentiated carcinomas (Fig. 2, *b*) and the remaining four were adenocarcinomas. One of the adenocarcinomas was a grade 3 lesion and the others were grade 4 carcinomas, one being a mucus-producing adenocarcinoma (Case 10).

The extent of direct invasion of the parietal pleura and the intercostal muscle bundles by the tumors was studied carefully. The carcinomas in 13 cases had invaded extensively into the intercostal muscle bundles surrounding the intercostal vessels and nerves (Fig. 3). As already mentioned, invasion of

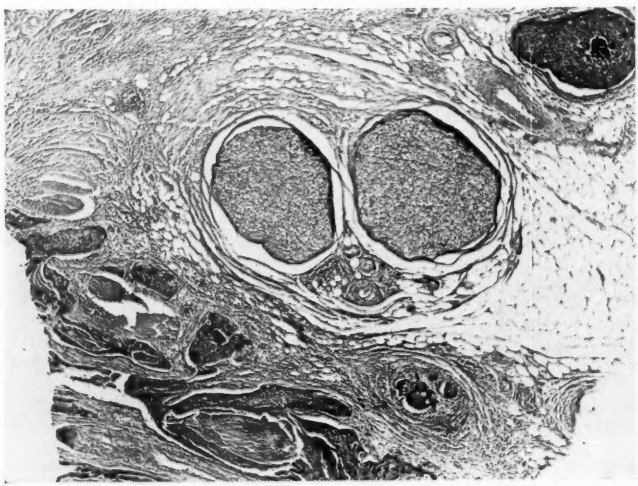

Fig. 3.—(Case 8). Carcinomatous invasion around the intercostal nerves (hematoxylin and eosin; ×30).

a vertebra also was present in one instance (Case 7) in addition to the intercostal involvement, and another patient (Case 14) had the additional complication of involvement of the brachial plexus. The intercostal muscle bundles were not involved in three instances (Cases 2, 9, and 13), the invasion including only the parietal pleura (Fig. 2, *b*).

The regional lymph nodes were involved in 3 patients (Cases 8, 9, and 16); only one involved node was detected in Cases 8 and 9, whereas massive invasion of the hilar nodes was present in Case 16.

LENGTH OF SURVIVAL AFTER OPERATION AND ITS RELATIONSHIP TO EXTENT OF THE LESION

Follow-up data were obtained in all 16 cases. Ten patients have died. The longest survival time in these 10 patients was 14 months (Case 4) and the shortest was 21 days (Case 7). The average length of life after operation in this group was 7 months. The intercostal muscle bundles and the structures in them, including nerves and blood vessels, were invaded by carcinoma in all 10 of these patients. Included were those 4 patients (Cases 1, 5, 8, and 10) in whom erosion of ribs was noted in the preoperative thoracic roentgenograms. The two patients (Cases 7 and 14) in whom carcinoma was known to have been left survived for only 21 and 50 days, respectively.

Four patients (Cases 2, 9, 12, and 13) who have been followed for more than one year were still living at the time of inquiry and had no evidence of recurrence. The survival times in these 4 patients ranged from 13 months to 6½ years (see table). As already noted, 3 of these patients (Cases 2, 9, and 13) had involvement of the parietal pleura only. The intercostal muscle bundles were invaded by carcinoma in Case 12. The regional nodes were not involved in Cases 2, 12, and 13, whereas one hilar node was found to be the site of metastasis in Case 9.

Two other patients (Cases 15 and 16) were living at the time of inquiry but they have been followed less than a year.

COMMENT

Many authors, such as Jones and co-workers, agree that the 3-year and 5-year survivals after pneumonectomy or lobectomy for bronchogenic carcinoma have been largely among the patients who had peripheral lesions without involvement of the thoracic wall. Jones and associates have been performing lobectomy for peripheral lesions that did not show nodal involvement and were away from the fissures. Twenty (69 per cent) of their 29 patients who underwent lobectomy were living 3 years after operation. On the other hand, their over-all 3-year survival rate was 28 per cent.

Therefore, it is reasonable to believe that peripheral bronchogenic carcinoma with involvement of the thoracic wall may be associated with a favorable prognosis if there has been no spread by the lymphatic system or blood vessels of the thoracic wall. Naturally, definite conclusions cannot be drawn from study of this small series but the available data appear to support this theory. Only 2 patients in our series have survived more than 2 years after operation; neither of these patients presented any evidence of invasion into the intercostal muscle bundles and most likely there was no permeation of the lymphatic system or the blood vessels of the thoracic wall. Symptomatically, this type of an early lesion was associated with malaise of 4 months' duration in one patient and with pain in the thoracic wall of 2 weeks' duration in the other.

Extension into the intercostal muscle bundles and invasion around the intercostal nerves and vessels apparently indicate a poor prognosis. Our patients with such advanced lesions usually had noted pain in the thoracic wall for

several months before the diagnosis was established. Roentgenographic evidence of erosion of ribs appeared to be an extremely grave prognostic sign. The disease progressed with great rapidity in the 2 patients in whom removal of the entire carcinoma was impossible.

These 16 patients comprise a highly selected group. For this reason, no effort has been made to establish any figures for the incidence of involvement of the thoracic wall or for its operative treatment. It is also unjustifiable on the same grounds to attempt to draw any conclusions as to the dominant type of cell in the lesions invading the thoracic wall. The only significant finding in this regard was the absence of small-cell carcinomas in this group. It has been pointed out previously by McBurney and associates that the small-cell carcinoma is usually a central lesion.

Involvement by carcinoma of the hilar nodes in only 3 of our 16 cases is a noticeably low incidence, in view of the fact that Carlisle and co-workers found nodal involvement in 43.7 per cent of their cases of squamous-cell carcinoma. Ochsner and associates noted nodal metastasis in 60 per cent of their patients in whom resection was done. This incidence was 70 per cent in Rienhoff's series. The low incidence of nodal involvement in the peripheral lesions in our series appears to support the concept that lobectomy may be a satisfactory procedure in carefully selected cases.[4, 7]

SUMMARY AND CONCLUSIONS

A study has been made at the Mayo Clinic of 16 cases of bronchogenic carcinoma in which involvement of the thoracic wall was present. Pneumonectomy was performed in seven instances and lobectomy in nine. The involved part of the thoracic wall was removed simultaneously in all cases.

The most prominent symptom was pain in the thoracic wall. No cases of Pancoast's syndrome were present in this series.

Six of the sixteen tumors were grade 4 squamous-cell carcinomas, six were large-cell undifferentiated carcinomas and four were high-grade adenocarcinomas. The intercostal muscle bundles and the structures in them were invaded in all but three instances.

Follow-up data were obtained in all cases. Ten patients have died from their disease. Two of the 6 surviving patients have lived more than 2 years (2⅓ years and 6½ years, respectively) after operation. The carcinoma did not extend into the intercostal muscle bundles in either of these 2 patients. The third patient who had involvement of the parietal pleura only was living 13 months after operation.

This study suggests that either pneumonectomy or lobectomy done in association with resection of the thoracic wall for bronchogenic carcinoma can be a curative procedure if the involvement of the thoracic wall is limited to the parietal pleura.

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RADIATION MANAGEMENT OF APICAL LUNG TUMORS

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APICAL lung tumors are separated from other lung lesions for this discussion because their clinical and radiotherapeutic behavior is different.

Terminology.—Our criteria of apical tumors are:

1. The lesion should arise from the apex.

2. The "apex" is denoted in its topographical meaning and not as the anatomic apical segment. The topographical "apex" implies a peripheral origin and location of the lesion to which the peculiarities of apical lesion are due; the apical segment might also include more central, larger bronchus branches.

3. The lesion should show an early tendency to peripheral invasion, toward the extrapulmonary tissues, because of the relatively small dimension and peripheral locality of the apex.

If apical and subapical areas are involved it may be difficult to differentiate which area was affected primarily. If clinical or radiologic signs of chest-wall involvement (pleural thickening, Horner's syndrome, shoulder-arm pains, rib destruction) developed relatively early, we can assume that the lesion arose from the apex. In late stages, however, nonapical tumors may also extend secondarily into the apical area and produce the same symptoms and signs as apical tumors. Occasionally, the classification may be clarified by available earlier x-ray films. Fig. 1, *A* shows an extensive upper lobe lesion which could be classified only after the patient's chest films of previous years were obtained. These films (Fig. 1, *B* and *C*) clearly localized the initial lesion in the apex. This was interpreted for two and one-half years as recurrent old tuberculosis as the patient had only moderate, vague complaints before the correct diagnosis was made. We classify doubtful cases in the lobar carcinoma group, for example, the case shown in Fig. 2. A massive tumor shadow occupied the apex, with moderate subapical extension. However, dense peribronchial strands leading to the hilum, and the absence of rib destruction and Horner's syndrome aroused suspicion of a central-bronchial origin; bronchoscopy confirmed it by showing the primary lesion close to the bronchial orifice.

4. If subapical areas are also involved, these should be secondary extensions from the apex.

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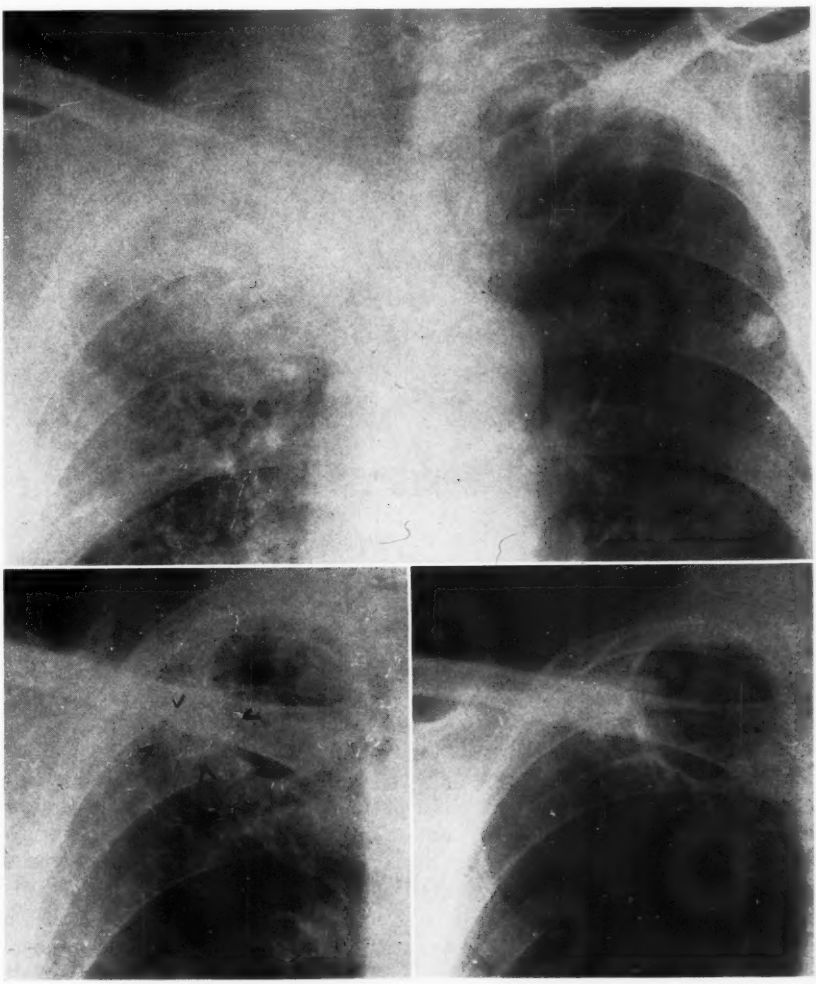
Pathology.—The term, "Pancoast tumor," or "superior pulmonary sulcus tumor," is no longer regarded as a clinical or pathologic entity,[2, 4, 5, 13, 14, 19, 22, 23] and we no longer use it. We still use the term "Pancoast syndrome,"[20] described also by Hare (in 1838), Donald (in 1850), and Tobias[24] (in 1932), to denote the clinical manifestations of a far-advanced chest-wall involvement

A.



B. *C.*

Fig. 1.—Case 11. *A*, At time of diagnosis: extensive lesion of RUL (involving hilar and mediastinal nodes, and second and third ribs).

B, Thirty months before diagnosis: hazelnut-sized soft shadow in apex. Primary apical lung lesion (interpreted as recurrence of old tuberculosis).

C, Sixteen months before diagnosis: apical shadow increased (still diagnosed as tuberculous lesion).

in the apical region which invades the sympathetic ganglion chain in the paravertebral area. Thereby, the lesion may arise initially from any part of the apical or subapical areas of the lung or of the extrapulmonary tissues, and its histopathology may be very diversified.[2, 18, 19, 22]

In the literature, a few authentic cases[1, 3, 7, 10] of primary epithelial tumors of the thoracic inlet without lung involvement are recorded. These tumors arose from unknown extrapulmonary tissues, but the great majority of those described originate from terminal lung bronchioles.[1, 13, 15, 23] As far as we could determine in our cases by radiologic or surgical means, or on surgical pathologic specimens, the lung was always involved with pleural or peripulmonary extensions at the time of diagnosis. It is in general impracticable to determine whether the pulmonary or the extrapulmonary lesion is primary. However, the x-ray films, the histopathology, where available, and the clinical course favored the assumption that all tumors in our cases arose

Fig. 2.—Tumor mass in left apical and subapical area; dense peribronchial strands leads to the hilum. Classified as lobar, not as apical, peripheral lung carcinoma. Primary lesion was found close to the upper bronchus orifice.

from the apical lung parenchyma. One can assume an early stage not yet involving the chest wall (see Fig. 1), as well as an intermediate stage with chest-wall involvement but without bony and neural invasion. This is illustrated by Fig. 3. The available minifilm of June, 1949, was negative, but that of June, 1952 (Fig. 3, *A*), shows a density in the center of the apex, presumably in the lung, which was unnoticed because of rib superimposition. Diagnosis was made only two and one-half years later, in November, 1954, also by a mass survey film (Fig. 3, *B*). The patient still had no clinical signs or symptoms, except for occasional vague shoulder discomfort, which was revealed only by direct questioning. Roentgenograms and surgical resection showed a pulmonary mass with involvement of posterior and mediastinal pulmonary walls which could not be entirely removed. The cases illustrated by Figs. 1 and 3 indicate how long the tumor may be present before the diagnosis is made.

We attempted subdivision of the disease according to its extension into the following *stages.*

Stage 1. Pulmonary involvement only.

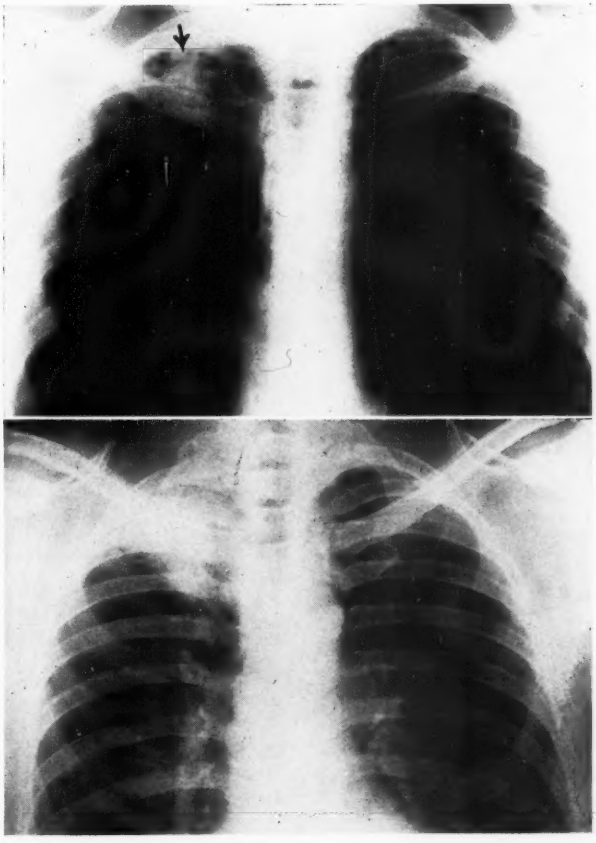
Stage 2. Involvement of chest-wall soft tissues, including mediastinum, only radiologically or surgically detectable (no Horner's syndrome or bony destruction).

Stage 3. Extension to bones: (a) ribs, and (b) vertebrae.

Stage 4. Paravertebral extension with sympathetic neural involvement, indicated by Horner's syndrome.

Stage 5. Regional or distant metastases.

A.



B.

Fig. 3.—Case 9. *A*, Density in right apex, unnoticed in mass survey by rib superimposition.
B, Thirty months later: intrapulmonary mass with pleural involvement, verified by incomplete segmental resection.

It is not essential that all symptoms of the Pancoast syndrome be present in early stages.[9, 19, 23] The most constant and earliest indication is pain, occurring early after involvement of the parietal pleura, or intercostal, or brachial plexus nerve branches. The Horner syndrome may appear later after encroachment of the sympathetic chain, but it may remain absent if the same lesions extends laterally and avoids the paravertebral area. Wasting of the muscles of the hand, secondary to disuse or to neural involvement and emphasized by Pancoast, was usually a late finding in our cases. Rib destruction was also absent initially in some cases. However, the absent signs may develop later, after the diagnosis has been made, following the extension of the tumor.

MATERIAL

Our apical tumor patients were irradiated, in part, (a) by conventional x-rays and, in part, (b) by high energy x-rays of the betatron.

TABLE I. DATA OF CASES TREATED BY CONVENTIONAL X-RAYS

CASE-AGE	SMOKING	HISTOLOGY	SYMPTOMS	WEIGHT LOSS	SIDE	STAGE	INVOLVEMENT				DURATION (MONTHS)	
							PLEURAL CHEST WALL	BONY	NEURAL	METASTASES	FIRST SYMPTOM TO FIRST DIAGNOSIS	FIRST DIAGNOSIS TO FIRST TREATMENT
1. M. C. 49	?	Carcinoma, minimal evidence (aspiration)	Chest pain, cough	++	L	2	+	0	0	0	4	3
2. L. J. 64	++	Class IV cells (aspiration)	Chest pain, arm pain, cough, dyspnea	++	R	3a	+	+	0	0	4	3
3. D. T. 66	++	Poorly differentiated carcinoma	Chest pain, cough, hemoptysis	?	R	3b	+	+	0	0	17	2
4. S. A. 49	?	Carcinoma	Shoulder pain	?	L	4	+	0	+	0	?	1
5. K. C. 63	+	?	Shoulder pain, chest pain, cough, dyspnea	?	R	4	+	+	+	0	4	1
6. C. W. 62	++	Poorly differentiated carcinoma	Chest pain, cough, neck mass	?	R	5	?	0	0	+ neck	10 days	2
7. M. J. 46	+	Undifferentiated carcinoma	Shoulder pain, chest pain, leg numbness, hoarseness, neck mass	++	L	5	+	0	+	+ neck	5	10 days
8. M. R. 54	?	Carcinoma simplex	Shoulder pain, neck nodes	?	L	5	+	+	+	+ neck	12	1

The reports of 8 cases, treated by *conventional x-rays* over the past twelve years, were found in our files and details are given in Table I. We treated 10 patients in six years by 22.5 million volt *betatron x-rays*, the details of which are outlined in Table II.

TABLE II. DATA OF CASES TREATED BY 22.5 MEV BETATRON X-RAYS

CASE-AGE	SMOKING	HISTOLOGY	SYMPTOMS	WEIGHT LOSS (LBS.)	SIDE	STAGE	INVOLVEMENT				DURATION (MONTH)	
							PLEURAL CHEST WALL	BONY	NEURAL	METASTASES	FIRST SYMPTOM TO FIRST DIAGNOSIS	FIRST DIAGNOSIS TO FIRST IRRADIATION
9. N. G. 55	++	Epidermoid carcinoma with adenocarcinoma qualities	Slight arm pain, slight cough	12	R	2	+	0	0	0	30 (minifilm)	3
10. M. B. 63	+	Aspiration: +	Chest pain, disabled, weak	20	R	3a	+	+	±	0	10	2
11. B. C. 67	++	Class IV cells	Chest pain, cough, hemoptysis, weak, disabled	20	R	3a	+	+	0	0	32 (Tbc. diagnosis)	1
12. A. R. 63	++	?	Back pain, cough, weakness, disabled	25	R	3b	+	+	±	0	21	3
13. M. M. 60	++	Epidermoid carcinoma	Arm pains, numb fingers, neck fullness	?	L	4	+	+	+	0	6	5½
14. B. H. 57	+	?	Shoulder pain, neck swelling, vena cava syndrome, disabled	12	R	4	+	+	+	0	5 (Osteop. treatment)	3 wks.
15. S. J. 58	?	Adenocarcinoma	Shoulder-arm pain	23	R	5	+	0	0	+ brain hilus	6 (Chiropr. treatment)	2
16. L. C. 60	++	Poorly differentiated carcinoma	Chest pain, cough dyspnea	30	R	5	+	+	?	+ skin	12 (Tbc. diagnosis)	1
17. B. A. 51	?	Adenocarcinoma	Shoulder-arm pains, anorexia, weak, disabled, neck nodes	9	L	5	+	+	+	+ neck	8	1
18. C. E. 65	0	Epidermoid carcinoma	Shoulder-arm pain, hemoptysis, neck swelling, weak, disabled	?	L	5	+	+	?	+ skin	2	3

The 18 cases are further summarized as follows: Table III shows that all patients were white males, except one white female (Case 18). Of 13 patients with recorded smoking habits, 8 were heavy smokers (over one package of cigarettes daily), 4 moderate smokers (one package or less per day); the one female did not smoke.

TABLE III. DISTRIBUTION BY AGE, SEX, RACE, SMOKING HABITS

	CONVENTIONAL X-RAY THERAPY	BETATRON THERAPY	TOTAL
Age	46-66 yrs. (average 56.6)	51-67 yrs. (average 59.9)	46-67 yrs. (average 58.4)
Sex			
Male	8	9	17
Female	-	1	1
Race			
White	8	10	18
Negro	-	-	-
Smoking habits			
Heavy	3	5	8
Moderate	2	2	4
Nonsmoker	-	1	1
Unknown	3	2	5

Table IV shows that the histopathologic distribution of the 10 cases determined is similar to that of other bronchogenic cancers.

TABLE IV. HISTOPATHOLOGY

	CONVENTIONAL X-RAY THERAPY	BETATRON THERAPY	TOTAL
Epidermoid carcinoma	-	2	2
Epidermoid carcinoma with adenocarcinoma qualities	-	1	1
Adenocarcinoma	-	2	2
Undifferentiated—poorly differentiated carcinoma	3	1	4
Simplex carcinoma	1	-	1
Undetermined type or not examined	4	4	8

Table V shows the presenting symptoms at the time of irradiation. Shoulder, arm, and chest pains were the most frequent complaints, but weight loss, weakness, and cough also occurred frequently. Atrophy of the muscles of the hand was usually either absent, or mild when first seen, and thus not recorded in several histories.

TABLE V. SYMPTOMS AT BEGINNING OF RADIATION

SYMPTOM	CONVENTIONAL X-RAY THERAPY	BETATRON THERAPY	TOTAL
Shoulder-arm pain	5	6	11
Chest pain	5	4	9
Numbness in fingers or toes	1	1	2
Weight loss	3	8	11
Weakness, disability	2	6	8
Cough	4	4	8
Dyspnea	2	1	3
Hemoptysis	1	2	3
Hoarseness	1	-	1

Table VI shows the anatomic involvement at the time irradiation was started.

TABLE VI. ANATOMIC EXTENT OF TUMOR AT BEGINNING OF RADIATION

	CONVENTIONAL X-RAY THERAPY	BETATRON THERAPY	TOTAL
Side			
Right apex	4	7	11
Left apex	4	3	7
Stage			
1	-	-	-
2	1	1	2
3a	1	2	3 } 5
3b	1	1	2 }
4	2	2	4
5	3	4	7
Extension			
Pulmonary cavitation	2	1	3
Bones: Ribs	4	8	12
Vertebrae	2	5	7
Sympathetic chain	4	3 + 2	7 + 2
Supraclavicular protrusion	1	2	3
Engorged veins	-	1	1
Neck node	3	2	5
Hilar, or mediastinal node	1	3	4
Distant metastasis	-	skin 2 }	3
		brain 1 }	

The right side was more frequently involved than the left side (11:7).

Using our staging (p. 499), no patient in Stage 1 was treated (without extrapulmonary involvement) because the diagnosis was not made earlier. In only 2 cases did reinspection of prediagnostic x-rays suggest the occurrence of apical lesion without extrapulmonary extension. Two patients were treated who did not have bony or sympathetic nerve or metastatic involvement, but who had surgically verified chest-wall invasion (Stage 2). In 5 cases, bony destruction was present without signs of sympathetic nerve or metastatic involvement (Stage 3). In 4 cases, Horner's syndrome was present without metastasis (Stage 4), and in 7 cases, metastases were already found either in the regional nodes or in distant areas (Stage 5).

According to anatomic structures, pleural involvement was assumed in 16 cases and could not be diagnosed in 2 cases, although both patients already had metastases. Destroyed ribs were found in 12 cases, vertebral erosion in 7 cases. The rib involvement started frequently as a pressure erosion, marked radiologically by notching with maintenance, later with loss of cortical layers (Figs. 4 and 5). Rib lesions may be demonstrated frequently better and earlier on oblique films (Fig. 5), particularly in the paravertebral area. Actual invasion of the bone by tumor was probably a secondary process (Fig. 6, *A-C*), but the two stages could not be classified separately. Sympathetic chain involvement was diagnosed in 9 cases (in 7, complete Horner's syndrome, in 2, only anisocoria). Supraclavicular protrusion of the apical tumor was seen in 3 cases, in one case with engorgement of the neck and chest-wall veins (Fig. 7, *A-D*). Metastatic neck nodes were found in 5 patients, mediastinal or hilar nodes in 4, and distant metastasis in 3 (2 skin, 1 brain).

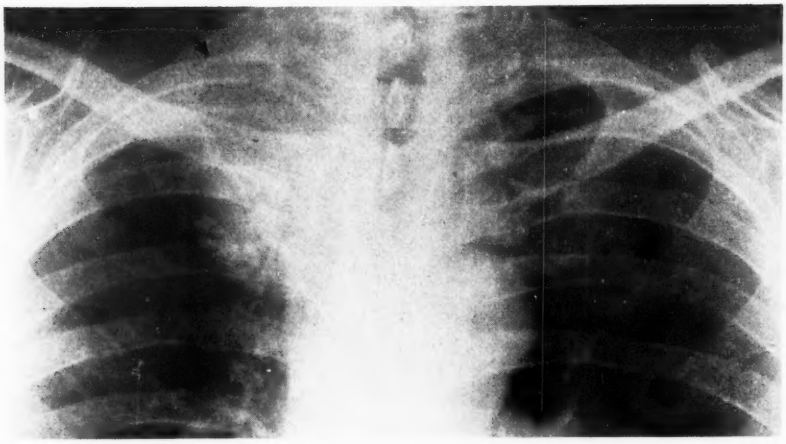


Fig. 4.—Case 5. Notching of the first rib, with visible cortical layer.

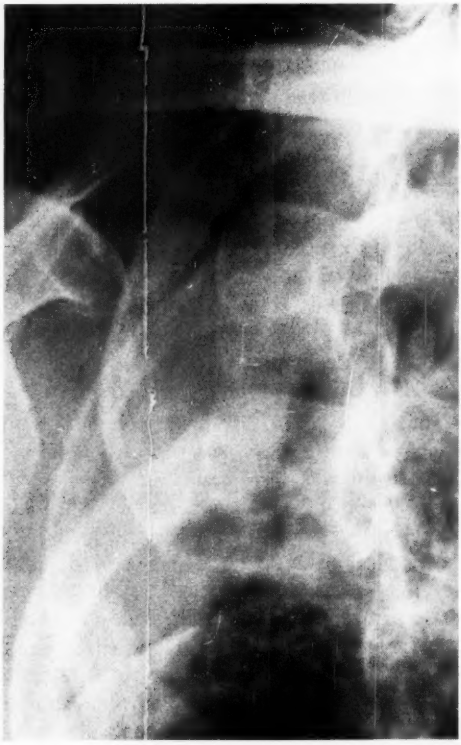


Fig. 5.—Case 16. Local indentation of first rib with loss of cortical layer. Diffuse erosion of the posterior lower margin of the second rib, with partially maintained cortical layer.

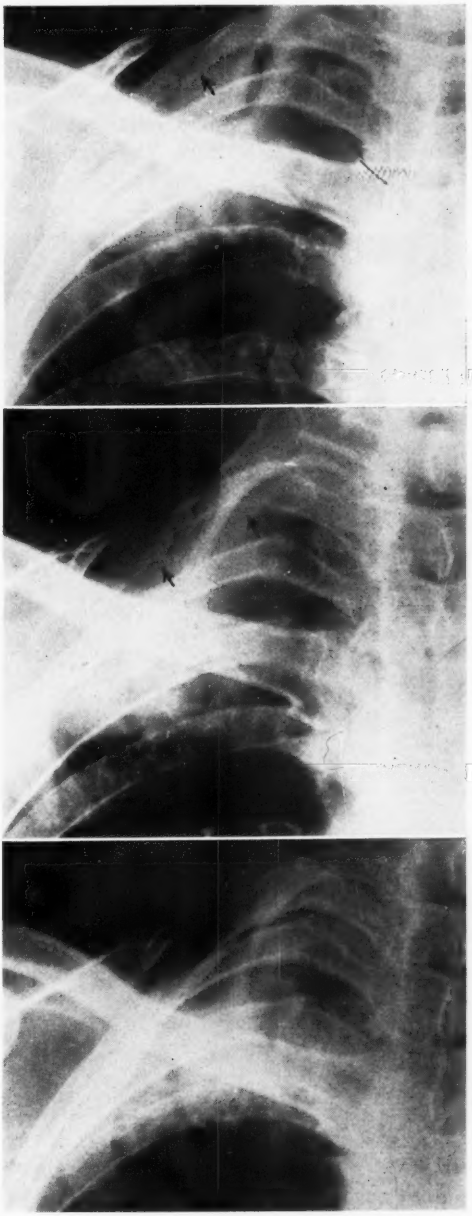
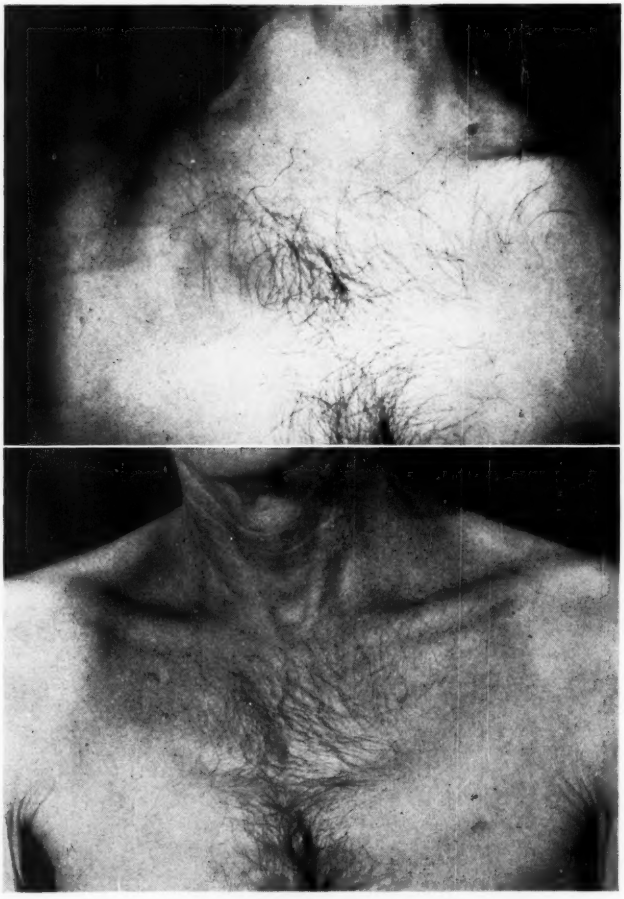


Fig. 6.—Case 10. *A*, Flat indentation of the second rib with largely maintained cortical outline. Beginning bony invasion laterally. *B*, Eight weeks later: cortical layer still present medially. In the lengthened lateral segment of the erosion actual bone invasion occurred with destruction of both cortical contours. *C*, Sixteen months after betatron irradiation: restored rib, radiation fibrosis in the lung.

Table VII shows the duration of symptoms or signs prior to diagnosis, and the interval from diagnosis to the first irradiation. These data show that the former interval in several cases was as long as one year, up to 32 months, in average over 10 months; that the latter interval was as much as 5½ months, in average 2 months. The prediagnostic delay was due in part to faulty diagnosis and therapy, preradiation delay referable mainly to intervening surgical procedures (resection, exploration). Thus, the irradiation was started in average 12 months after onset.

A.



B.

Fig. 7.—Case 14. *A*, Supraclavicular protrusion of apical tumor, engorgement of neck and chest-wall veins. *B*, After completing betatron irradiation: regression of protrusion and of engorgement. Deepened supraclavicular fossa. Moderate pigmentation.

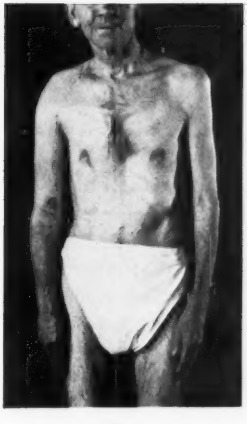
RADIOTHERAPY

The technique and results of irradiation are considered separately for groups treated with conventional x-rays and with betatron x-rays.

TABLE VII. DURATION OF SYMPTOMS OR SIGNS BEFORE DIAGNOSIS AND BEFORE FIRST IRRADIATION

	CONVENTIONAL X-RAY THERAPY		BETATRON THERAPY		TOTAL	
	MONTHS	AVERAGE (MONTHS)	MONTHS	AVERAGE (MONTHS)	MONTHS	AVERAGE (MONTHS)
From first symptom to first diagnosis	½-17	7.0	½-32	12.3	½-32	10.1
From diagnosis to first irradiation	¼-3	1.7	1-5½	2.2	¼-5½	2.0

C.



D.

Fig. 7.—Cont'd. *C*, After 1½ years: supraclavicular fossa is filled up by firm fixed fibrotic tissue (radiation fibrosis); in the center biopsy scar. Residual venous dilatations visualized by infrared photography.

D, After 3 years: retraction of right upper thorax by pulmonary fibrosis; muscular atrophy of the shoulder-arm and paresis of the hand due to damage of brachial plexus by cervical radiation fibrosis.

Technique.—

1. *Conventional x-rays:* In the 12-year period, different techniques were used by different therapeutists in the 8 cases. One incompletely reviewed case is apparently cured for over 11 years (see later). This case was irradiated with 400 kv. 2.75 mm. copper half value layer, in 3 fields of 10 by 10 cm., 50 cm. target skin distance, 9,150 r total air dose in 60 days. A rough estimation of the dose to the tumor center was 5,300 r.

Among the other 7 cases, 200 kv., 0.9 mm. copper half value layer was used in one case, 215 kv., 1.05 mm. copper half value layer in one; 400 kv., 2.75 mm. copper half value layer in 3 cases, 400 kv. with grid in 2 cases. The calculated or retrospectively estimated tumor dose varied between 4,200 and 7,500 r, the over-all treatment time between 30 and 129 days.

2. *Betatron therapy:* Ten cases were treated with 22,500,000 volt x-rays of the betatron with multiportal technique, 4 to 8 ports being employed. After adequate experiences and systematic isodose curve analysis, we reduced the number of fields, usually to 5. The field size varied, according to the shape and dimensions of the patient and to the extent of the lesion, from an 8 to 10 cm. square portal to a 15 cm. circle. The over-all treatment time varied between 26 and 44 days. The shorter times were for technical reasons in our early experimental period but the majority were within 40 to 43 days. The total dose given to the center of the tumor was 8,000 to 11,000 betatron r; the dose in the tumor as found by isodose curves varied between +4 and −8 per cent. We included the paravertebral area and the lateral side of the vertebrae in the fields as much as possible and we tried to avoid the spinal cord. However, we now feel that a greater part of the vertebrae should be included, if paravertebral invasion can be assumed, because some residuals and recurrences were found at autopsies in the prevertebral and cord areas in spite of healed pulmonary and peripulmonary lesions. Moreover, the radiologic examinations in some other cases showed progression of the vertebral lesion, with regression of the pulmonary lesions. We observed spinal symptoms in 2 cases after radiation. In 4 cases, following completion of a full radiation course with inadequate response, or with recurring symptoms, and after some hesitation, we gave a second series of treatments, taking into account the risk of radiation damage. Our philosophy was that it would be preferable for the patient to die from possible late radiation damage after an additional 1 to 2 years of useful life, than in a few short months from uncontrolled disease. One of these patients had a useful survival for 18 months after the second series, and died in 10 weeks of spinal symptoms (see Fig. 11). One other patient survived 26 months after the second series, with 24 months of useful life, dying of spinal and cerebral metastases (see Fig. 12). Both had a good response for 12 and 4 months, respectively, after the first radiation series. In the other 2 cases, in which response to the first radiation course was slight, or of short duration, the second series was also a failure.

RESULTS

A. *Survival.—*

1. *Conventional x-ray treatment:* As mentioned before, one case (Stage 2)

is living and well after 11 years. The aspiration biopsy was reported as "carcinoma, diagnosis based on minimal evidence." Histologic slides and preoperative x-rays are not available for review, so we accept the diagnosis with caution. However, the characteristic description of the pulmonary and inoperable extrapulmonary invasion from the exploratory surgical findings leave little doubt of the diagnosis. We regard this case as one of the rare, but known curiosities with exceptional healing tendency. The other 7 cases had survivals after the first irradiation of 3 to 9 months, with an average of 6 months. The average survival according to the stages was 7.5 months for Stages 3 and 4, and 4 months for Stage 5.

The survival time after the first symptoms in 6 of the cases was 5 to 26 months, averaging 14 months, and after diagnosis 5 to 12 months, with an average of 7.4 months.

TABLE VIII. DURATION OF LIFE

STAGE	GROUP	NUMBER OF CASES	LIVING AND WELL	DECEASED	SURVIVAL AFTER ONSET MONTHS	SURVIVAL AFTER ONSET AVERAGE	SURVIVAL AFTER DIAGNOSIS MONTHS	SURVIVAL AFTER DIAGNOSIS AVERAGE	SURVIVAL AFTER RADIATION MONTHS	SURVIVAL AFTER RADIATION AVERAGE
2-4	Conventional x-ray treatment	5	1	4	11-26	17.7	7-12	9.3	6-9	7.5
2-4	Betatron x-ray treatment	6	2	4	29-54	43.6	19-49	33.1	17-48	32.4
5	Conventional x-ray treatment	3	-	3	5-17	10.7	4.5-5	4.8	3-5	4.0
5	Betatron x-ray treatment	4	-	4	8-20	15.7	6-10	8.7	3-8	6.7
Total	Conventional x-ray treatment	8	1 (11 yrs)	7	5-26	14.0	5-12	7.4	3-9	6.0
Total	Betatron x-ray treatment	10	2 (11, 27 months after treatment)	8	8-54	29.7	6-49	21.0	3-48	19.6
Literature						12.0 (nonirradiated, 9.7; irradiated, 14)		6.0		6.0

2. *Betatron therapy:* Of the 10 patients treated with irradiation, 2 are living and well over 11 and 27 months, respectively, since the first irradiation, 43 and 60 months since the first symptoms.

The 8 deceased patients survived 3 to 48 months, with an average survival of 19.6 months after the first irradiation, 29.7 since the first symptoms. As to the staging, the survival after the first irradiation was 17 to 48 months, with an average of 32.4 months in Stages 3 and 4; and 3 to 8 months, averaging 6.7 months, in Stage 5 with regional or distant metastasis. This big difference in survival corroborates the old experience that if one lymphatic area is invaded, seeding is usually already present in other lymphatic pathways as well.

As noted, the survival time in Stages 2 to 4 was much longer in the betatron treated cases, averaging 32.4 months, against 7.5 months in the conventional x-ray cases. In Stage 5, this difference was only moderate, 6.7 months versus 4 months. It seems that the survival time does not vary appreciably for Stage 5 patients regardless of whether treated or untreated, or how irradiated.

These data of survival are summarized in Table VIII.

The therapeutic data of the treated patients concerning dosage, radiation effects, and final outcome are comprised in Tables IX and X.

TABLE IX. THERAPEUTIC DATA OF CASES TREATED BY CONVENTIONAL X-RAYS

CASE	RADIATION C. TUMOR DOSE R IN DAYS	SURVIVAL AFTER ONSET (MONTHS)	SURVIVAL AFTER FIRST RADIATION (MONTHS)	EFFECT CLINICAL	EFFECT X-RAY	REMARKS
1	5300 r 65 days	11 yrs.	11 yrs.	Cured	Cured	Explored: inoperable chest wall and mediastinal invasion, living and well over 11 yrs.
2	7500 r 45 days	16	9	All symptoms moderately improved	Decreased tumor, cavity, rib regeneration	Cavitation in tumor, perforated to chest wall
3	4700 grid 129 days	26	7	0	0	Cavity in tumor
4	4200 r 39 days	?	8	0	Some tumor decrease	Incomplete segmental resection, died of frontal lobotomy
5	4200 r 33 days	11	6	0	0	
6	4400 r grid 30 days	5	3	Slight improvement, decreased neck mass, subjectively improved	?	Died of heart and prostatic complications
7	6500 r 127 days	10	5	0	?	Also hilar nodes irradiated
8	5800 r 59 days	7	4	0	0	Died of spinal and brain metastases

B. *Palliation.*—

The criteria of results include not only curability or prolonged survival time but also palliative effects. Important in regard to palliation are the ways in which radiation helps the patient live with his disease during his remaining life. Significant accomplishments of this type consist of a decrease of suffering, a postponement and shortening of the terminal stage, ability to return to a useful family, social, and professional life for any length of time thereby getting the benefit of doubt, and acquisition of a hopeful psychologic attitude to life during a limited life span in cases where prolonged life cannot be expected to result.

1. *Conventional x-ray cases:* Of the 7 deceased patients, 5 were failures, showing no significant change in progression of the disease. Two cases improved temporarily. In one case, moderate regression of the complaints and of neck nodes was observed, but the patient died of complications of cardiac and prostatic disease. In the other case with gross vertebral erosions, the palliation was marked, all symptoms significantly improved for about 7 months, the pulmonary cavitation which also penetrated the chest wall decreased, and destroyed ribs partially regenerated (Fib. 8, *A-D*), but the patient died 9 months after the first treatment, following a rapid preterminal downhill course of 4 weeks.

2. *Betatron treated cases:* Of the 10 cases, 2 are alive in practically asymptomatic conditions. Of the 8 deceased patients, the palliation in one was insignificant, whereas in all other 7 cases it was very marked. These patients became asymptomatic or showed very marked improvement so that they could return to a useful life of 16 to 44 months in Stages 3 and 4 but only for 1 to 3 months in Stage 5. In Stage 5, life was not significantly prolonged, but the relief of suffering, even though temporary, was so striking that we would not deny this assistance to the patients.

The first and most frequently observed effect was the relief of incapacitating *pains in shoulder,* arm, and hand. This effect was observed at times as early as the second day of irradiation, leading us at first to believe in some cases, for instance, in Case 10, that it was a psychologic one, until its objectivity was proved by continuity of gradual pain relief. This pain practically subsided in 5 of our 6 cases, but in one case the relief was only temporary and moderate.

The *chest pain* subsided somewhat later, usually parallel to the radiologic regression of the lesions, and in all 5 cases where it was a presenting symptom.

The relief of pain was probably in great part the reason that 5 of our 6 weakened, disabled patients, some brought in by wheel chair, shortly regained their strength, became self dependent, could again use their arms, and could resume their normal life completely or with some limitations for variable intervals according to their further course.

Cough, hemoptysis, dyspnea also subsided, if present, parallel with the tumor regression.

Weight gain was a rather objective indicator of tumor regression. At times the original weight was exceeded. Recurring weight loss was usually a premonitory sign of recurrence, metastasis, or postradiative complication.

TABLE X. THERAPEUTIC DATA OF CASES TREATED BY 22.5 MEV BETATRON X-RAYS

CASE	RADIATION C. TUMOR DOSE R IN DAYS	SURVIVAL AFTER ONSET (MONTHS)	SURVIVAL AFTER FIRST RADIATION (MONTHS)	EFFECT CLINICAL	EFFECT X-RAY	USEFUL LIFE (MONTHS)	TERMINAL STAGE (MONTHS)	REMARKS
9	8,400 r 42 days	43	11	Asymptomatic, 20 lbs. gain	--	11 Living and well	--	Diagnosis by mass survey, incomplete resection
10	9,100 r 31 days	29	17	Asymptomatic 15 lbs. gain	Tumor fibrosis, rib regeneration	16	16 days	Died of heart symptoms, cardiac involvement?
11	8,000 r 44 days	60	27	Asymptomatic	Lung fibrosis, rib regeneration	27 Living and well	--	Extensive UL lesion to hilum. Posttreatment: pulmonary fibrosis with retraction, intermittent dysphagia
12	I. 10,000 r 40 days II. 7,600 r 24 days	54	33	I. Asymptomatic II. Asymptomatic	I. Bony regeneration II. Vertebral regeneration	I. 12 II. 18	10 weeks	Herpes zoster after first treatment, died of spinal symptoms
13	I. 10,000 r 41 days II. 9.000 r 43 days	37½	31½	I. Asymptomatic II. Asymptomatic	I. Lung tumor disappeared II. Rib. vertebral recalcification	I. 4 II. 24	2	Treatment after 2 resections; posttreatment: neck induration, disturbed arm function Autopsy: No pulmonary tumor, perivertebral involvment, spinal and cerebral metastases

14	11,000 r 26 days	54	48	Asymptomatic, temporary	Tumor fibrosed, rib regeneration	44	2 weeks	Posttreatment: neck induration, disturbed arm function; died after alcoholic avitaminosis with rapid cachexia and dysphagia, no tumor evidence
15	9,400 r 38 days	15	7	Pains gone, weight gain, diplopia	Tumor fibrosed, skull metastases	3	3	Autopsy: no tumor in thorax, cerebral metastases
16	I. 8,030 r 40 days II. 4,000 r 24 days	22	9	I. Almost asymptomatic II. 0	I. Cavity gone, tumor fibrosed	2	?	Cavitation of tumor, died of vertebral-spinal involvement
17	I. 9,650 r 39 days II. 6,500 r 19 days	18	8	I. Slight decrease pains II. 0	0	4 weeks	3	Autopsy: fibrotic apex with small residual tumor nodules, died of generalized metastases
18	10,000 r 43 days	8	3	Almost asymptomatic	Lung tumor regressed, bony lesions progressed.	2	1	Exploratory operation, died of generalized metastases

C. *Anatomic Effects.*—

The objective therapeutic effects observed clinically or radiologically were as follows:

1. The supraclavicular neck swelling, due to protrusion of the tumor filled apex, usually started to regress after a few treatments, and disappeared by the time irradiation was completed (see Fig. 7).

2. Metastatic neck nodes usually also regressed.

3. Engorged neck and chest-wall veins, present in one of our cases, rapidly regressed (see Fig. 7).

4. The pulmonary tumor shadow on x-ray films gradually regressed and was replaced slowly by fibrosis, later by homogeneous atelectatic induration (see below). In 2 of our 3 autopsied cases, residual tumor was not found in the lung; and in the third patient, who died shortly after completing radiation, marked local regression was seen, with only small residual tumor nodules remaining in the lung. Occasionally regression of pulmonary cavitation was observed (Fig. 9, *A-C*); however, our clinical results in cases with necrotic tumor cavitations were not favorable.

5. A sensitive objective sign of tumor regression was regeneration of destroyed bones (ribs and vertebrae) (Figs. 6, 10, *A* and *B*), starting a few weeks after irradiation, also shown in figures of our earlier papers.[11, 12] Understandably, vertebral involvement impairs the prognosis. However, it is remarkable that very extensive vertebral destruction also may be favorably influenced by radiation for longer times (Fig. 11, *A-D*). On the contrary, a progressing or newly appearing bony lesion was positive proof of progressive or recurrent disease (Fig. 12, *D*). Partial recalcifications were observed also in such cases in which the tumor regressed in this area, but active lesion was still present in distant parts of the lung or paravertebral area. However, if the regeneration was complete, this occurrence was less probable. A proof of recurrence is also the appearance of tumor mass in the prevertebral soft tissues (Fig. 12, *E*) on lateral or oblique neck films.

6. Regression of the Horner sign was not seen in any of our cases, including those with seemingly healed thoracic lesions, probably because destroyed nerve fibers did not regenerate. Appearance, or increase of Horner syndrome could be observed occasionally during the progression of the disease.

Radiation reactions and damages: Some patients had to pay a price for the clinical relief obtained in the form of radiation damage.

Direct radiation reactions were insignificant. The *skin reaction* never exceeded the stage of hyperemia, followed by slight pigmentation or slight scaling which was marked only in cases of short over-all treatment time. In the other cases it was mild.

Direct reactions of subcutaneous tissue were not observed. Complaints related to acute lung or bronchial reactions were not seen. In cases where the mediastinum had to be included in the radiation fields, hoarseness of short duration occurred in one case by tracheal reaction, and mild dysphagia due to esophageal mucositis in 2 cases. Systemic reactions were insignificant,

except for one case with mild radiation sickness, and another case with moderate radiation sickness where large fields (15 cm.) had to be used, both cases responded well to Dramamine.

In one case with vertebral erosion, herpes zoster developed after radiation, corresponding to the irradiated spinal segment.

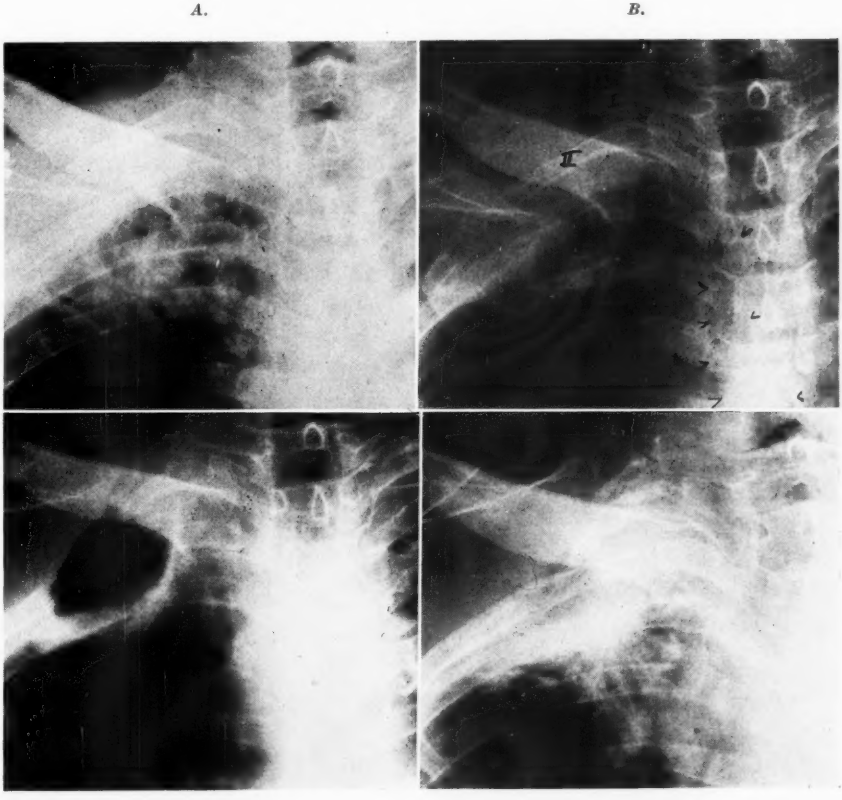


Fig. 8.—Case 2. *A*, Cavitations in apical tumor. Destroyed first and second ribs. *B*, Bucky film. Gross erosions mainly of the third, fourth, and fifth thoracic vertebrae. *C*, Three months after x-ray therapy: tumor diminished, cavitation penetrates the chest wall.

D, Eight months after irradiation: decreased cavity, partially regenerated ribs. Retracted upper lobe.

More significant were effects of *late damage*. These occurred because the patient lived long enough for these to occur and because of the high tumor and tissue dose, which was greater than in the usual series of treatments, exceeding the "tolerance" dose, especially in patients given a second treatment course.

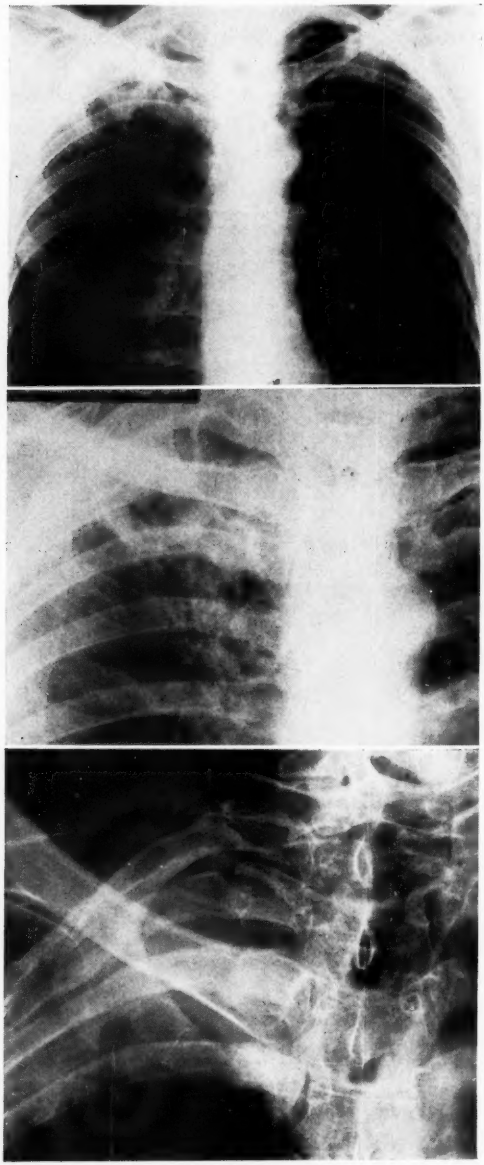


Fig. 9.—Case 16. *A*, Cavitation in right apex. Medially air-bleb. Diagnosed previously as tuberculosis. Rib lesions shown in Fig. 5.

B, Two months later, before irradiation: cavity enlarged, progression of rib destructions (not shown).

C, Six months after irradiation: cavity disappeared. Probably radiation lung fibrosis. Largely recalcified ribs. Recent bony involvement of T_2.

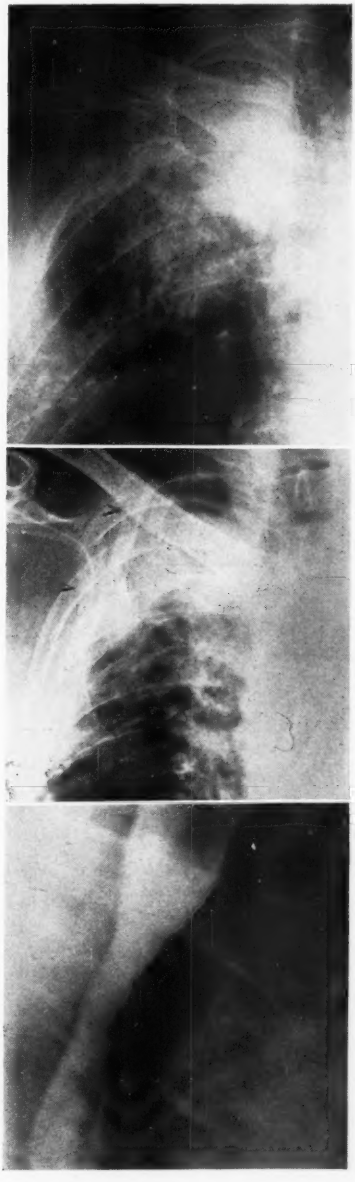
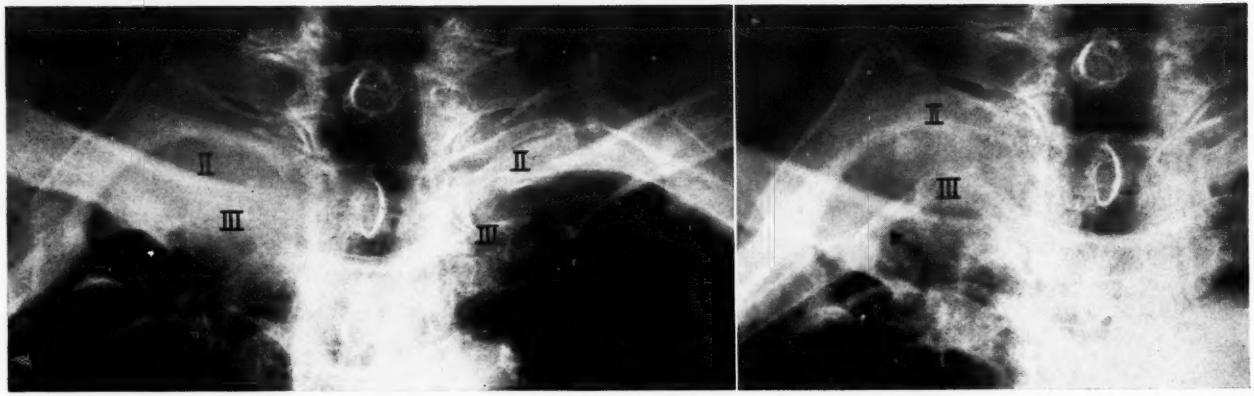


Fig. 10.—Case 11. *A*, Advanced lung lesion. Destroyed second and third ribs.
B, Fourteen months after betatron irradiation: regenerated ribs (>). Radiation fibrosis of lung.
C, Moderate esophageal narrowing in the irradiated area, causing intermittent dysphagia.



A. *B.*

Fig. 11.—Case 12. *A*, Lateral margin of T_2 body eroded. T_2 and T_3 transverse processes are very decalcified. Second and third ribs destroyed.

B, After betatron irradiation: T_2 margin more dense, better demarcated. T_2 and T_3 transverse processes recalcified, second and third ribs largely regenerated.

C.

D.

Fig. 11.—Cont'd. *C*, Recurrence after one year of asymptomatic condition: involvement of T_1, T_2, and T_3 transverse processes; body defect of T_2 body, erosion of T_3.
D, After second series of betatron irradiation: recalcification of destroyed vertebrae, transverse processes, and ribs.

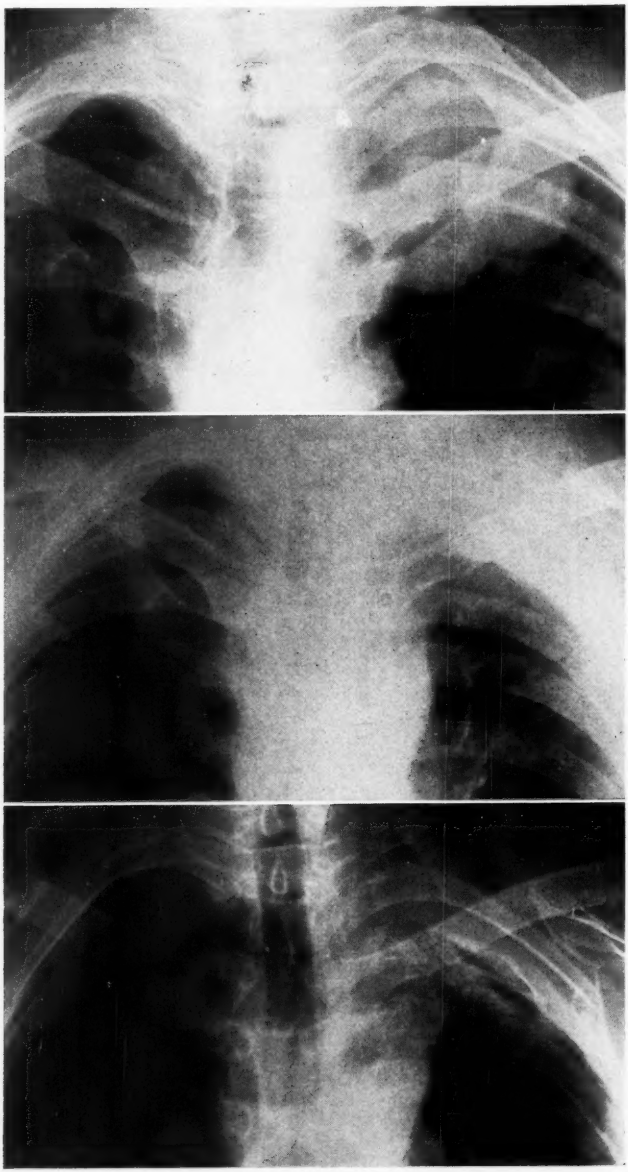


Fig. 12.—Case 13. *A*, Intrapulmonary tumor with chest-wall invasion, proved by resection.

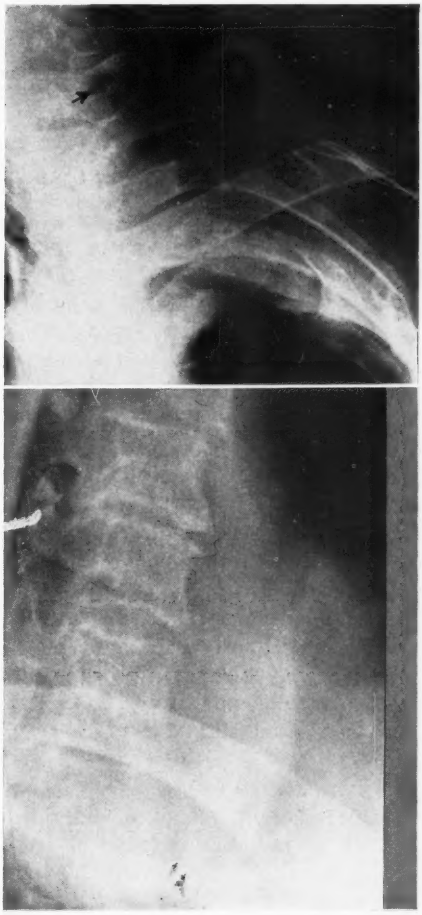
B, Recurrence after two surgeries. (Erosion of T_2 margin, not shown.)

C, After two series of betatron irradiation: fibrosis, retraction of irradiated lung. No evidence of bony involvement.

Late skin damage consisting of marked atrophy or telangiectasia was not seen. In some cases the skin was fixed to the indurated subcutaneous tissues.

Damage of greater clinical significance occurred in the subcutaneous tissues of the supraclavicular area. After tumor regression, this fossa was usually rather shrunken, with atrophic muscles due to disuse and to radiation

D.



E.

Fig. 12.—Cont'd. *D*, Clinical relapse: osteolytic lesion in T_1 pedicle area. *E*, Mass in prevertebral tissues at the same level (proved by autopsy).

fibrosis. After approximately 1½ to 2 years, the fossa filled up gradually by formation of immobile woody-firm scar tissue, fixed to the skin and to the periosteum, giving the appearance of a frozen infiltration (see Fig. 7, *C*). Biopsies and autopsies showed that this consisted only of fibrotic scar tissue.

This tissue sometimes later surrounded and compressed the brachial vessels and nerve plexus, causing first coolness and other circulatory disturbances in the arm (see Fig. 7, *D*), followed by paresthesias and paresis in the fingers, and limitations in shoulder movement. The discomfort was accompanied by varying degrees of aches usually controlled by varying doses of Empirin. Due to the clumsy use of his arm, one of our patients fractured his upper arm twice in rapid sequence through falls. Disturbed arm function occurred in 3 patients but, in one of these, autopsy disclosed a spinal metastasis as additional cause of the ulnar paralysis. Occasionally narrowing of the prevertebral soft tissues by radiation fibrosis was observed on lateral neck films.

The radiation damage next in importance was that of the *esophageal wall*, with late radiation mucositis and moderate narrowing causing dysphagia. We observed this in 2 cases. The narrowing, confirmed by x-ray examination and by endoscopy, was not significant enough to require dilatation or gastrostomy (see Fig. 10, *C*). In one case, the dysphagia was elicited 3 to 4 times yearly on a psychosomatic basis, and disappeared in 2 to 3 days by change of environment. In the other case, a few weeks after avitaminotic stomatitis (which healed promptly by vitamin medication) apathy and weakness appeared about 4 weeks before his death. One week later dysphagia developed, with the previously mentioned moderate x-ray and endoscopy findings which accounted for reduced food intake. The weakness and lethargic condition progressed until his quiet death, without signs of recurrence or metastases. How much of this malnutrition was due to his alcoholism or to dysphagia could not be analyzed, because he was not hospitalized, but the dysphagia possibly aggravated and accelerated the fate of the patient.

Clinical *bone damage* was not seen, although in one autopsy case the clavicle did show sclerotic changes in the spongiosa.

The irradiated *lung segment* showed radiologically (Figs. 10, *B*, and 12, *C*), and at autopsies in all cases, atelectatic and fibrotic collapse, with shrinking, followed by retraction of the apical thorax and mediastinum. Autopsies showed adhesion of the collapsed segments to the chest wall. The functional loss of the segment caused no clinical symptoms. In some cases moderate local discomfort was the chief complaint, due probably to the adhesions and retraction.

The possibility of *postradiation transverse myelitis* was considered clinically in 2 cases. In one of these, it remained undecided whether the spinal symptoms were produced by radiation myelitis or by neoplastic spinal involvement. However, vertebral bony involvement, clinical similarity to the second case, and inconclusive but suspicious myelogram favor the latter etiology. In the second case, autopsy revealed spinal involvement by tumor, without radiation damage of the spinal cord.

COMMENT

The literature is very pessimistic about the fate of these patients. The average survival after onset is listed by various authors[17, 22, 23] as 10 to 14 months. According to Moersch and associates,[19] the majority die within 6 months after the first examination. All agree in general that the apical tumor

is radioresistant, that radiation offers no benefit at all, or only rarely a very slight short-lived relief and diminution of tumor shadow.[5, 8, 10, 16, 17, 22] In some cases, the symptoms were rather aggravated.[6, 16] Only on a few cases was a palliation of several months described,[6, 14, 21] without significant prolongation of life. However, Eschbach and Finsterbusch[6] compiled 71 cases from the literature and 15 cases of their own, and found that, in 37 nonirradiated cases, the average survival from onset was 9.7 months, in 43 irradiated cases, 14 months. From 40 irradiated cases, 25 had no effects. In 15 cases, improvement was observed, represented in all by pain relief, in 6 by improved general condition, in 4 by decrease of the tumor, and in 2 by recalcification of destroyed ribs. Their data are complicated by inclusion of several cases of nonapical lung tumors, penetrating the chest wall. In recent years our institution has published reports of cases[11, 12] of good radiation effects, with regeneration of destroyed ribs, and recently Quick and Richmond[21] also described one case with recalcified ribs.

The comparison of the data presented in our paper with the literature shows that the survival time of our cases treated by conventional x-rays conforms to those cited in the literature, except for one random case, cured for 11 years. Of the betatron treated cases, 2 are living and well for 43 and 60 months after the onset; the deceased patients survived the onset by an average of 29.7 months, in Stages 2 to 4 by an average of 43.6 months (literature: 12 months). Their average survival after the first irradiation was 19.6 months, in Stages 2 to 4, 32.4 months (literature: 6 months[19]). In Stage 5, with metastases, the survival was not prolonged significantly; however, a good palliation was achieved in all but one case. Thus, radiation results regarding survival and palliation are far better in our betatron group than in our conventional x-ray cases, or than in those described in the literature. We do not believe that the biologic effect of the high energy radiation on the tumor is qualitatively different. The better betatron results should be attributed rather (1) to the higher tumor dose, (2) to the more homogeneous dose distribution in the tumor, and (3) to the more accurate and careful treatment planning and positioning. We believe that if the same care were exerted in treatment planning and technique with conventional x-ray therapy, this also could achieve some degree of results.

As seen, the histopathology of the apical tumors is as diversified as the other bronchogenic cancers, and they do not form a separate pathologic entity.

The response to radiation in Stage 5 is similar to other lung carcinomas but is much better in the earlier stages of the apical tumors. This can be explained by a combination of several factors, related to the peripheral localization and smaller volume of the apex. First, the lesion reaches the pleura and chest wall easier and elicits pain earlier, with a consequently earlier diagnosis. Second, the tumor extends peripherally with greater ease, the central lymphatics are involved later, and the mediastinal nodes less frequently. Therefore, local radiation can be more successful. Finally, the irradiated lung volume is more limited, thus the beams can avoid vital mediastinal organs as well as the opposite lung. Thus, radiation is better tolerated than in other localizations.

SUMMARY

1. The apical lung tumors do not form a separate pathologic entity, but a topographic distinction is made because, due to the particular peripheral location, their clinical and radiotherapeutic behavior is different from that of other lung cancers.

2. An attempt is made to subdivide the cases in 5 stages, dependent upon the degree of involvement.

3. Eighteen cases were irradiated at the University of Illinois College of Medicine, 8 with conventional x-rays, 10 with betatron x-rays.

4. The cases are reviewed and analyzed from diagnostic and therapeutic points of view.

5. The betatron radiation excelled by far the conventional x-rays in therapeutic effect, based upon comparisons within our own material as well as with the data in the literature. However, conventional x-ray therapy is also expected to achieve better results if similar care will be exerted in treatment planning and technique.

6. The conventional x-ray case figures show one cured for 11 years, regarded as an exceptional curiosity in radiosensitivity. In the other cases, the results conform to the data of the literature.

7. The 10 betatron case figures show 2 living and well, 11 and 27 months after the radiation. The deceased patients survived from the onset an average of 29.7 months, in Stages 2 to 4, 43.6 months (literature: 12 months); from the first irradiation an average of 19.6 months, in Stages 2 to 4, 32.4 months (literature: 6 months). In Stage 5 (with regional or distant metastasis) the prolongation of life was not significant, but the palliative effect was striking, in fact much better than implied in the literature.

8. The radiation effect on subjective symptoms (pain, etc.) was manifest in their disappearance or marked decrease for variable durations, and in significant reduction of the patient's suffering, and duration of the terminal stage.

9. The objective radiation effect could be followed clinically or radiologically by reduction or disappearance of the tumor, by recalcification of destroyed costal or vertebral bony structures, by gain in weight and strength, by resumed and prolonged useful life, and by disappearance or regression of the tumor in autopsied cases.

10. It is worth while to irradiate all cases of apical tumors, in Stages 2 to 4 for significantly prolonged periods of useful life and possible cure, in Stage 5 as a palliative aid to living during the remaining life of the patient. The diagnosis is rarely made in Stage 1, in which surgery would be academically the therapy of choice.

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CORONARY AND CAROTID ARTERY PERFUSION DURING TOTAL BY-PASS OF THE HEART

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AS a result of the early and continued interest of Gibbon[6-8] and the recent contributions of Andreasen,[1] Lillehei,[2] and Kirklin,[3] and their associates, repair of ventricular septal defects has become a reality. Recently, Warden and his co-workers[4] reported continuous arterial reservoir perfusion during cardiac by-pass. The following method of perfusing arterialized blood from a reservoir during cardiac by-pass has been used experimentally for the past year and a half. With this technique, only the brain and heart receive blood during the period of by-pass and this paper reports the results of 121 experiments.

METHOD

Dogs weighing from 4 to 30 Kg. were used. Anesthesia was induced with 2½ per cent Pentothal and an endotracheal tube was inserted. The animals were maintained with ether and oxygen administered with intermittent positive pressure. The femoral artery and vein were cannulated for pressure measurements. A small number of animals were not cooled. The remaining dogs were cooled by immersion in a water and ice bath (3° to 4° C.). The animals were removed from the bath when the rectal temperature was 3° or 4° C. higher than the desired final temperature, to allow for the usual drop. Sterile technique was not employed.

A right thoracotomy was performed through the fourth intercostal space. A heavy silk ligature was passed around each vena cava. The right brachial (subclavian) artery was isolated and ligatures were passed around it and all of its branches, except for the internal mammary, which was cannulated for the measurement of blood pressure during the period of by-pass. The mediastinal pleura was opened and ligatures passed around the left brachial (subclavian) artery and the aorta. A polyethylene catheter was inserted into the right brachial artery in the chest or the right carotid artery in the neck for subsequent perfusion of the aortic arch. This catheter was connected through a finger pump to a reservoir of heparinized, oxygenated blood. Another catheter was inserted into the superior vena cava through the azygos vein.

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The actual period of by-pass was carried out as follows: The superior vena cava was occluded and its blood siphoned into a graduated cylinder. The left brachial artery was temporarily occluded and the branches of the right also, if it was not used for perfusion. The inferior vena cava was next occluded. The aorta was cross-clamped immediately distal to the origin of the left subclavian artery and perfusion begun (Fig. 1). An incision, 4 to 5 cm. in length, was made in the right ventricle and the coronary sinus blood continuously aspirated (Fig. 2). In all animals, the right ventricle was open for 5½ to 31 minutes. The ventricle was then closed, the lungs were inflated, normal circulation was restored, and perfusion was discontinued.

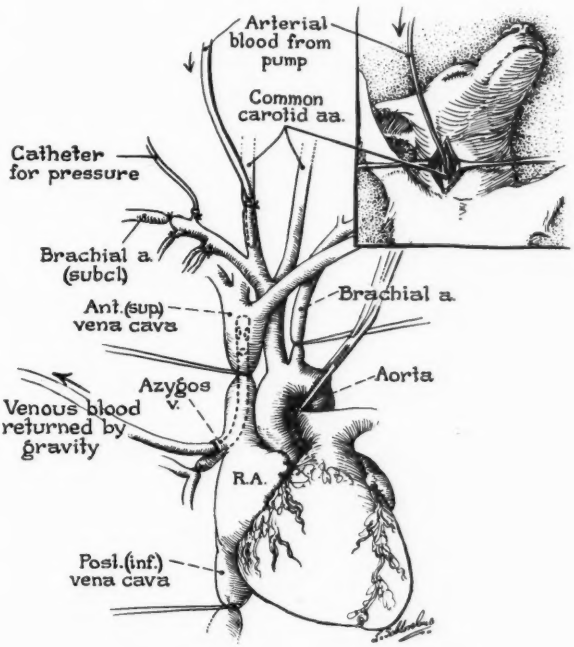

Fig. 1.—Schematic representation of the perfusion system.

The arterial blood used for perfusion was obtained from the femoral arteries of donor dogs. The blood was collected in bottles containing 40 mg. of heparin per liter of blood. No determinations of blood incompatibility were made and frequently pooled blood from 6 or 7 dogs was used. The blood was pumped from a 2-liter graduate after filtration through commercial recipient sets.

In most animals, the sum of the blood collected from the superior cava and from the right ventricle was 150 to 250 ml. greater than the amount perfused during the period of by-pass. This deficit was replaced slowly with an in-

travenous transfusion of citrated blood after restoration of the circulation. The animals were given protamine sulfate (1 mg./Kg.) to counteract the heparin that remained following perfusion.

RESULTS

GROUP I. This technique was used to by-pass the heart in 11 dogs under normothermic conditions. The aorta and brachial arteries were occluded for 8 to 18 minutes in these animals, and the right ventricle was open for 5½ to 17 minutes. Four of the animals were chronic survivors. The period of occlusion

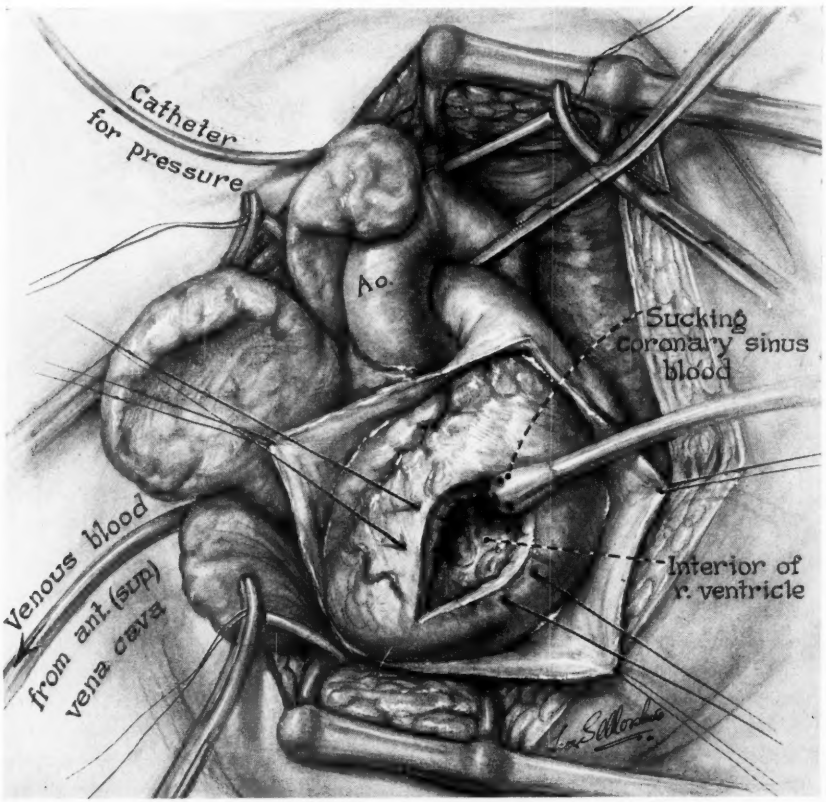

Fig. 2.—Operative exposure obtained during the period of complete by-pass.

in these surviving animals was 8½, 12, 14, and 18 minutes. Four of the dogs died 16 to 36 hours after surgery. The aorta and brachial arteries were clamped for 12 to 15 minutes, and all 4 dogs revealed evidence of cord damage with spastic paralysis of the hind legs. Three more animals died of postoperative bleeding within the first 24 hours.

GROUP II. Eighty-five dogs were cooled to a final rectal temperature of 25° to 30° C. The aorta was cross-clamped and both brachial arteries were occluded for 15 to 31 minutes. The right ventricle was open for 15 to 30 minutes. During the period of by-pass the blood pressure was maintained at 80 to 90 mm. Hg in most of the animals. Within a minute after stopping the perfusion and restoring the circulation, the blood pressure was usually above 100 mm. Hg. Thirty-six of the 85 dogs were chronic survivors. The remaining 49 died within the first 3 postoperative days. Six of the dogs died of cardiac arrest within a few minutes after perfusion and restoration of the circulation. In 6, the cause of death could not be determined at autopsy. In 10 animals, the cause of death was thought to be due to hemorrhage, and in 27 animals the cause of death was pulmonary congestion. This was confirmed by pathologic examination.

Twenty-one of the animals developed ventricular fibrillation during perfusion or after restoration of the circulation. It was possible to defibrillate all of the hearts, but only 7 of these 21 dogs were chronic survivors.

There were several points of interest in this group of animals. A record was kept of the amount of blood recovered from the superior vena cava, as compared to the amount recovered from the right ventricle. More blood was always recovered from the superior vena cava during the period of by-pass, but this varied considerably. The range was fairly evenly distributed between a ratio of 2 to 8 times as much recovered from the superior vena cava as from the right ventricle. In a few animals, 14 and even 18 times as much blood was perfused to the head as to the heart. There was no relationship between this ratio and survival.

In this group of animals, the amount of blood required to maintain a pressure of 80 to 100 mm. Hg during the period of by-pass was about 150 c.c. per minute in a 15 Kg. dog. There was a great deal of individual variation, however.

In 9 animals, immediately after opening the right ventricle, a Foley bag catheter was inserted into the pulmonary artery and the bag inflated. Through this catheter, blood was perfused into the lungs by gravity during the period of by-pass. Ten to 35 c.c. of blood were perfused per minute in a 10 to 15 Kg. dog. The survival rate in this group was no better than when only the head and heart were perfused; 5 of these 9 dogs died.

Twenty animals were digitalized prior to cardiac by-pass. Seven of the animals were digitalized by giving them 0.15 mg. of acetyl strophanthidin in three doses. The drug was given intravenously at 5-minute intervals so that the last injection was given just prior to by-pass. Nine of the dogs were given 1 mg. of Digoxin intravenously 4 hours prior to cooling, and another 0.5 mg. intravenously as the chest was being opened. The initial dose always made the dogs vomit. Of these 20 dogs, only 8 were chronic survivors, so that there was no increased survival among the dogs that received a digitalis preparation.

In 34 animals, the lungs were oxygenated with 100 per cent oxygen during the period of by-pass. Ether was not administered during this period. Of these

34 animals, 15 were long-term survivors. Therefore, the mortality in this group was about the same as the over-all mortality.

GROUP III. Twenty-five dogs were cooled to a final rectal temperature of 30° to 32° C. The aorta was clamped for 15 to 37 minutes, and the right ventricle was open for 15 to 31 minutes. In this group of animals, an attempt was made to apply the information gained in previous experiments. The lungs were deflated during the period of by-pass in this group. A blood pressure of 85 to 125 mm. Hg was maintained in all of these dogs during the period of by-pass. In this series of experiments, a 15 Kg. dog received approximately 175 c.c. of blood per minute to maintain this pressure during the period of by-pass. One minute after closing the ventricle the tie around the superior vena cava was released, allowing some of the superior vena caval blood to enter the right atrium. Over the period of the next minute to a minute and one-half, the catheter in the superior vena cava was gradually occluded, so that more blood returned to the right atrium. The ties around both brachial arteries were released. The pump was gradually slowed and the inferior caval constriction gradually released. Extreme care was taken to prevent overdistention of the right atrium. The aortic clamp was gradually released. None of the animals developed ventricular fibrillation during the period of by-pass or after restoration of the circulation. Four of the dogs died within 36 hours of pulmonary congestion, and the other 21 are long-term survivors.

DISCUSSION

Prior to using the method just described, total by-pass of the heart was accomplished by using a dispersion type oxygenator and a finger pump. In 27 animals, the entire animal was perfused and, in 14 more, the aorta was cross-clamped at the origin of the left brachial artery during the 15 to 30 minutes of by-pass and right ventriculotomy. A systolic pressure of 80 mm. Hg was usually maintained during this period. All of the dogs except one died of shock, bleeding, or pulmonary congestion.

Because of these discouraging results, it was felt that the survival rate could be improved if the heart and head alone were perfused. In this way, the pool of normal blood in the rest of the body would not enter the extracorporeal circuit. In the early experiments with carotid and coronary artery perfusion, the body temperature was not lowered. However, with the aorta and both brachial (subclavian) arteries clamped, cord damage frequently occurred with periods of occlusion of 12 to 15 minutes. Hypothermia was then employed, with rectal temperatures of 25° to 30° C., but usually closer to 25° than to 30° C. However, because of an incidence of ventricular fibrillation of 25 per cent, and because of a chronic survival rate of only 40 per cent, the animals were perfused at progressively higher temperatures.

In the course of the experiments, an attempt was made to evaluate certain factors. In some of the animals, the pulmonary artery was perfused with blood during the period of by-pass. Another group of animals was digitalized. In still another group of animals, the lungs were inflated during the period of

by-pass. None of these factors seemed to influence the survival rate. Several factors seemed to be important. The final rectal temperature should not be lower than 30° to 32° C. In this range, there was no ventricular fibrillation whereas in the animals perfused at a final rectal temperature of 25° to 30° C. there was a 25 per cent incidence of ventricular fibrillation.

It was important to maintain a blood pressure of at least 85 mm. Hg during the period of by-pass. Slow release of the inflow clamps over a period of 4 to 5 minutes and release of the aortic clamp over a period of ½ minute seemed important in order to maintain a good blood pressure and coronary circulation. Despite all of these precautions, however, 4 of the last 25 dogs died with pronounced pulmonary congestion within 48 hours after operation. In all of these animals, the right ventricle was open for 15 to 30 minutes. These results are similar to those reported by Shumway, Gliedman, and Lewis.[5] They perfused the coronary but not the carotid vessels after first cooling the animals to 25° C. They felt that in dogs it was safe to by-pass the heart for periods up to 20 minutes at this temperature with coronary perfusion of 50 to 85 c.c. per minute.

SUMMARY

A technique is described for cardiac by-pass in the dog permitting right ventriculotomy for 30 minutes. The head and heart are perfused with oxygenated, heparinized blood while the animal is maintained at a rectal temperature of 30° to 32° C. In the last group of 25 dogs, 21 are long-term survivors.

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INTRATHORACIC NEUROFIBROMA OF THE VAGUS NERVE ASSOCIATED WITH A DIAPHRAGMATIC HERNIA

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ALMOST unusual and completely unsuspected finding during the course of the repair of an esophageal hiatus hernia of the diaphragm was a large neurofibroma of the vagus nerve supplying the lower end of the esophagus.

In a review of their own series of 18 patients with intrathoracic neurofibroma and in 100 cases of others collected from the literature, Kent, Blades, Valle, and Graham[1] found no evidence of the vagus nerve as a point of origin of these nerve tumors. The infrequency of reports of neurofibromas of the intrathoracic as compared to the cervical portion of the vagus nerve suggests the rarity of this lesion. We have encountered only four such specific reports in the available literature. In 1943, Blades and Dugan[2] reported a case of multiple intrathoracic nerve fibromas, two of these originating in the left vagus nerve. In 1950, Parrella[3] reported a neurofibrosarcoma of the left vagus nerve which was located at the base of the neck and extended into the anterior superior mediastinum. In 1953, Gerbode and Marguiles[4] reported an interesting case of neurofibromatosis (von Recklinghausen's disease) with neurofibromas involving the left vagus nerve in the superior mediastinum. A year later, Gilbertsen and Lillehei[5] published a case report which concerned a 40-year-old man with bilateral cervicothoracic neurofibromas of the vagus nerves in the absence of neurofibromatosis. Physiologic observations were carried out to study the effect of removal of the tumors on the function of the cardiorespiratory systems. The usual site of intrathoracic nerve sheath tumors is in the posterior mediastinum with origin from the sympathetic chain or the intercostal nerves, rather than the vagus nerve.

CASE REPORT

Mrs. J. K. was first seen in 1944 when, at the age of 55, she was referred by her family physician with the diagnosis of pernicious anemia refractory to liver therapy. Signs and symptoms of anemia had then been present for one year and she had had five blood transfusions. Initial investigation, in 1944, revealed a normocytic hypochromic anemia, occult blood in the stools, normal gastric secretions, and an esophageal hiatus hernia (Figs. 1 and 2). On the diagnostic assumption that bleeding resulted from peptic erosion associated with the diaphragmatic hernia, treatment comprised modified Sippy management plus transfusions and iron. Although the therapeutic response was prompt, the patient refused to remain on medical measures after the acute symptoms subsided. During the following twelve years she had 15 major gastric hemorrhages. From 1947 on, operative repair of the hiatus hernia was recommended with increasing persistence because of the conviction that

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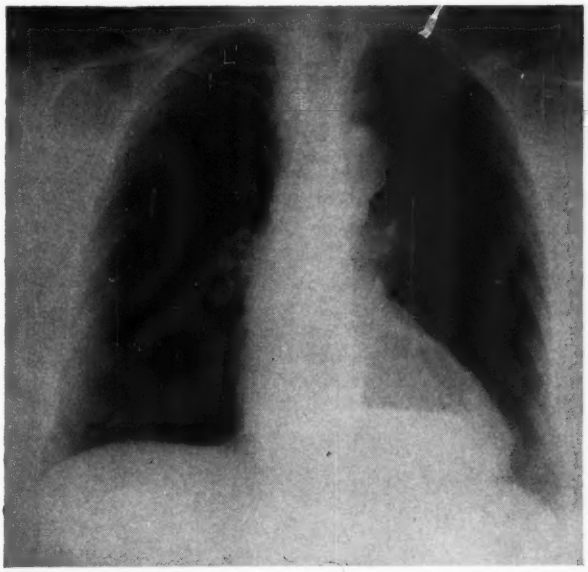

Fig. 1.—Roentgenogram of the chest demonstrates a fluid level in the intrathoracic portion of the stomach.

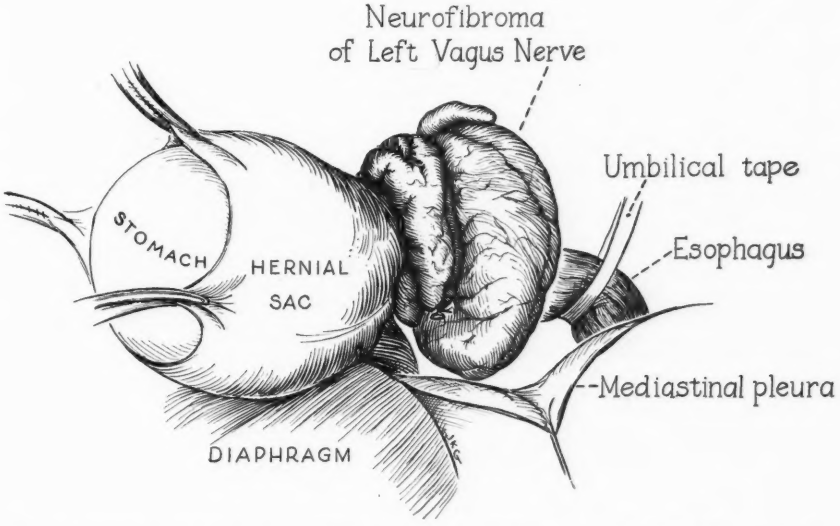

Fig. 2.—Drawing sketched at operation shows the relationship of the neurofibroma to the esophageal hiatus hernia.

peptic ulceration was occurring in this pouch although an ulcer crater could not be conclusively demonstrated fluoroscopically. Finally, after a particularly depleting episode of melena had been brought under control, the patient entered the Presbyterian Hospital, Chicago, on Aug. 31, 1955, for surgical treatment of a diaphragmatic hernia. It is of particular interest that the patient had never experienced abdominal or thoracic discomfort and had no digestive tract symptoms or signs other than those due to hemorrhage. There were no pregnancies. There was no history of trauma. Extreme obesity had been present for thirty years.

Examination on admission revealed a 66-year-old white woman, 65 inches in height, weighing 200 pounds. The blood pressure was 198/100 mm. Hg. Other physical findings were normal, as were the hemogram, urinalysis, electrocardiogram, and examination of the feces.

On Sept. 1, 1955, a left thoracotomy was performed through the bed of the resected seventh rib. Upon incising the mediastinal pleura, the preoperatively diagnosed esophageal hiatus hernia presented itself, as well as an unsuspected gray white, semilunar-shaped tumor which encircled the lower end of the esophagus. The origin of the tumor was a large trunk of the left vagus nerve. A frozen section identified this lesion as a neurofibroma.

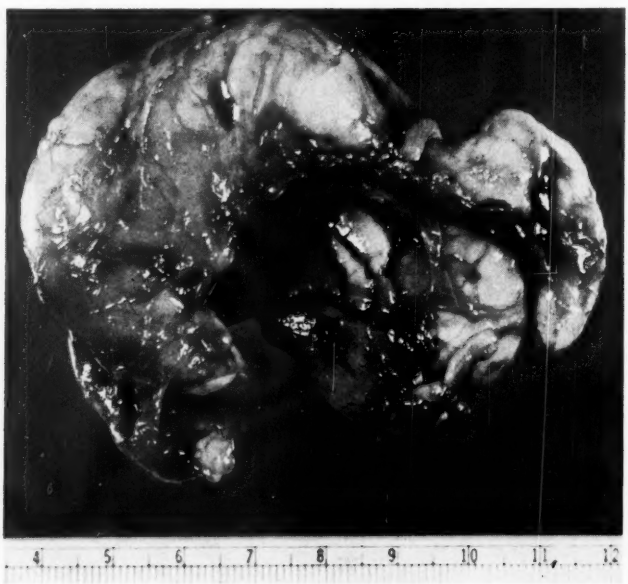

Fig. 3.—Photograph of the surgical specimen.

While resecting the tumor, a small segment of the anterior wall of the stomach was removed at the level of the cardio-esophageal junction. Exploration through the opening in the gastric pouch revealed no evidence of recent or old peptic ulceration nor any esophageal varices. In closure of the defect, an anastomosis was accomplished between the anterolateral wall of the esophagus and adjacent stomach. The excess portion of the hernial sac was excised. The esophageal hiatus was reconstructed with transplantation of the esophagus, anteriorly. After an uneventful postoperative course, the patient was discharged on the sixteenth postoperative day.

The patient was re-examined six weeks after operation. The wound was well healed. There were no particular complaints. Solid foods were swallowed without difficulty. At this time a roentgen examination of the esophagus and cardiac end of the stomach showed no evidence of stenosis, regurgitation, or ulceration. The stomach was confined to the peritoneal cavity.

Pathology—gross examination: The mediastinal tumor consisted of a somewhat circular mass of gray tissue, measuring 9.5 by 4.5 by 2.3 cm. (Fig. 3). The specimen weighed 60 Gm. On one side of the specimen there was a triangular section of gastric mucosa removed from the cardiac end of the stomach. On the inferior side of the specimen there was a portion of the left vagus nerve, measuring approximately 2.5 cm. in length. At one point there was evidence of bifurcation of the nerve.

Pathology—microscopic examination: The tumor was fibrous in character and consisted essentially of a proliferation of Schwannian cells. There was massive fibrous overgrowth with interspersed hyaline fibers, often arranged in concentric groups between interlacing bundles of elongated cells, having many of the features of fibrocytes. The tumor was relatively acellular in various places, the result of abundant collagenous deposits. The palisade arrangement of nuclei was not as conspicuous as in some neurogenic tumors (Fig. 4). In one section, there were several areas of calcification; in another section, at the level of the esophageal-gastric junction, an intense subacute inflammation underlying the stratified squamous epithelium was present; however, the gastric mucosa appeared normal. The histologic diagnosis was neurofibroma of the vagus nerve, subacute inflammation of the esophagus.

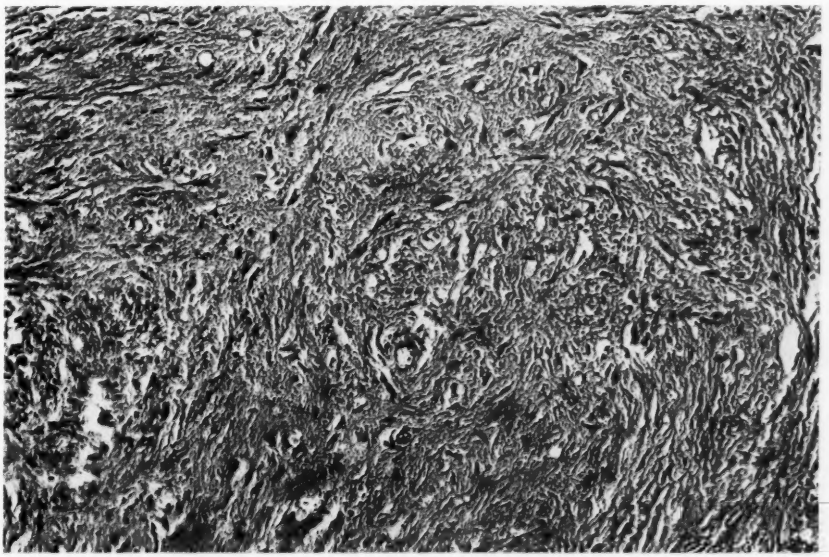

Fig. 4—Photomicrograph of the neurofibroma showing the nuclei of nerve cells disseminated through interlacing bundles of fibrous connective tissue.

DISCUSSION

Of salient interest in this case is the 12-year history of recurrent gastric hemorrhages, associated with a large esophageal hiatus hernia. The ready response of these bleeding episodes to acid-neutralizing therapy led to the belief

that evidences of peptic ulceration would be found at operation. The absence of such evidence or signs of venous thrombosis in the gastric pouch eliminates the mechanisms of hemorrhage considered most likely in this case. The inadequacy of present knowledge concerning this important complication, which has an incidence as high as 20 per cent among symptom-producing hiatus hernias, is further emphasized.[5]

Although symptoms from this hernia appeared at an age common for such lesions and in a very obese woman, no definite etiologic factors can be implicated. In spite of the intimate association of the vagus neurofibroma with the hernia, it seems unlikely that either one contributed to the development of the other. The finding of calcium in the tumor suggests the chronicity of this lesion and the 12-year presence of the hernia is a matter of radiologic record.

The operative procedure has produced an apparent cure. No deleterious effects have been observed to result from the resection of portions of the left vagus nerve and the neurofibroma.

Although there are a number of reports of nerve sheath tumors arising from the vagus nerve in the neck, this case is unusual in that only four other instances of intrathoracic tumors arising from the vagus nerve are found in the available literature.

CONCLUSIONS

The case is that of an elderly, obese, white woman in whom a large esophageal hiatus hernia had been repeatedly demonstrated for twelve years. Her symptoms over this long period were those of acute and chronic blood loss, presumably due to peptic erosion in the gastric pouch. Although numerous attempts had been made to bring the patient to surgery, it was only a particularly massive gastric hemorrhage that prompted the patient to consent to operative treatment.

At operation, a mediastinal tumor was found with its origin in the left vagus nerve innervating the lower end of the esophagus. Histologically, this resembled a neurofibroma. Repair of the esophageal hiatus hernia resulted in disappearance of symptoms. At the present time, seven months postoperatively, there are no complaints referrable to the surgery. The patient is swallowing solid foods as well as liquids without any difficulty. The anemia is corrected.

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VENTRICULAR FIBRILLATION

An Experimental Study Comparing Various Voltages and Durations of Electric Shock in Defibrillation of the Canine Heart

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THE use of electric shock is the accepted method for the treatment of ventricular fibrillation. The design of the electrical defibrillator is generally agreed upon but there is still disagreement as to the optimal voltage and duration of the shock. Kirby, Johnson, Engelberg, and Rovis[1] concluded that 220 volts for 1/10 second was uniformly more successful than lower voltages in defibrillating canine hearts. They felt that this voltage and duration of shock would not cause burns of the heart. Kay[2] and Mackay, Mooslin, and Leeds[3] have recommended lower voltages. Because of these conflicting results the following study was undertaken.

METHOD

Forty-eight mongrel dogs weighing 7 to 15 Kg. were anesthetized with intravenous thiopental sodium. An endotracheal tube was inserted and 100 per cent oxygen was administered by intermittent positive pressure through a demand valve. Using sterile technique, a left thoracotomy was performed through the fifth intercostal space and the pericardium was opened widely anterior to the phrenic nerve. Ventricular fibrillation was induced by passing a shock of 10 volts for ½ to ¾ second through the heart. The heart was allowed to fibrillate for four minutes during which time ventilation was continued. The heart was then massaged for two minutes at a rate of 40 to 50 times per minute and a single shock was then used in an attempt to defibrillate the heart. After defibrillation, the pericardium was left open and the chest was closed in layers. The dogs were sacrificed at intervals of two to twenty-six days and the hearts were examined.

The dogs were divided into four groups of twelve. In all four groups the heart was massaged for two minutes after the four-minute period of fibrillation. In the first group, defibrillation was attempted with a single shock of 130 volts at 1/10 second duration. In the second group, defibrillation was attempted with a single shock of 130 volts at ¼ second duration. The third group was treated with a single shock of 230 volts at 1/10 second. In the fourth group

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of animals, 230 volts at ¼ second was the initial shock. The failures in all groups were massaged for one minute and defibrillated wtih a single shock of either 230 volts at 1/10 second or 130 volts at ¼ second. Only the hearts of those dogs that had been defibrillated on the first attempt were examined for evidence of myocardial burns. Microscopic sections were taken from the sites where the paddles were applied.

The electrical defibrillator was that described by Kouwenhoven and Kay[4] with the addition of a 1-2 step-up isolation transformer and an electronic timing unit.* The timing unit permitted shocks of 1/10 and ¼ second with an accuracy of 1/100 second. The electrodes were made of brass rods, 6 mm. in diameter, with one end bent at a right angle to the axis of the rod. These rods were insulated with rubber tubing. A stainless steel disk, 45 mm. in diameter, was riveted to the bent end of each electrode. Plaster felt, 4 m.m. thick, was sewn to the disks. The disks were saturated with normal saline before being applied to the heart.

RESULTS

In the first group, a single shock of 130 volts for 1/10 second was effective in 8 of 12 animals (Table I). Ten of 12 animals, comprising the second group, were defibrillated with 130 volts for ¼ second. In the third group, 10 of 12 animals were defibrillated using 230 volts for 1/10 second. Two hundred and thirty volts for ¼ second was successful in 10 of 12 animals in the fourth group. A second shock of either 130 volts for ¼ second or 230 volts for 1/10 second was effective in the total of 10 dogs of the four groups in which fibrillation persisted after the first shock. Serial shocks were not employed in this study. Eight of the 48 animals died. Six of the 8 died within three days and were found to have bilateral pulmonary congestion. The remaining 2 animals died on the third and seventh postoperative days with overt evidence of brain damage. Their lungs were normal.

TABLE I

INITIAL SHOCK	TOTAL NUMBER OF DOGS	NUMBER OF HEARTS DEFIBRILLATED WITH INITIAL SHOCK	NUMBER OF MYOCARDIAL BURNS			
			MINIMAL	MODERATE	SEVERE	TOTAL
130 V 1/10 second	12	8	2	0	0	2
130 V ¼ second	12	10	1	3	0	4
230 V 1/10 second	12	10*	1	2	1	4
230 V ¼ second	12	10	5	2	2	9

*Sections of one heart were lost. Nine hearts were examined microscopically.

Of the 8 animals that died, 3 had required a second shock for defibrillation. These 3 animals died of pulmonary congestion and did not exhibit evidence of brain damage.

*Designed and constructed by the Instrument Section, National Institutes of Health, Bethesda, Md.

PATHOLOGIC OBSERVATIONS

Gross Findings.—Thirty-seven of the 38 hearts that were defibrillated with one shock were examined grossly and microscopically. They were weighed with the blood removed and a minimum of the great vessels remaining. The weights were between 61 and 125 Gm. Some of the hearts revealed minimal subepicardial hemorrhage that was attributed to massage.

There were no grossly visible burns of the hearts that were defibrillated with 130 volts at either 1/10 or ¼ second. Of the hearts defibrillated with 230 volts at 1/10 second, three of four burns were grossly visible, and of those hearts defibrillated with 230 volts at ¼ second, six of nine burns were visible grossly. The lesions were located on the right ventricle just below the right atrial appendage and at the apex of the left ventricle. These were the sites of application of the electrodes.

Microscopic Findings.—Two sections, stained with hematoxylin and eosin, were prepared from each of 37 hearts defibrillated with a single shock. The sections of the heart from the thirty-eighth dog, defibrillated with 230 volts at 1/10 second, were lost. The blocks were taken to include the entire ventricular wall at the point of contact of the electrodes.

The burns were divided into three groups according to the following criteria: The minimal lesions were less than one low-power field (10×) and located in either the outer or middle one third of the myocardium. The moderate lesions were larger than one low-power field and located in the outer one third of myocardium, or smaller than one low-power field and located in the inner one third of the myocardium. If the lesions were greater than one low-power field and located in either the middle or inner one third of the ventricular wall, they were classified as severe.

Two of the 8 hearts defibrillated with 130 volts at 1/10 second revealed microscopic burns. These were extremely small and peripheral in location. They were classified as minimal (Fig. 1*C*). Three of the 10 hearts defibrillated with 130 volts at ¼ second had burns that were moderate, and one other was minimal. Of the 10 hearts defibrillated with 230 volts at 1/10 second, 9 were examined, revealing four burns. There was an extensive severe burn involving the entire thickness of the right ventricle of one heart (Fig. 1*A*). Of the remaining lesions, two were moderate and one was minimal (Figs. 1, *D* and *E*). In the series of hearts defibrillated with 230 volts applied for ¼ second, 9 of 10 hearts showed myocardial burns of varying degree. Two lesions were classified as severe (Fig. 1*B*); two as moderate; and five as minimal (Fig. 1*F*).

DISCUSSION

Hooker, Kouwenhoven, and Langworthy[5] demonstrated that a current of less than 0.8 ampere will not stop fibrillation and a current greater than 0.8 ampere will not induce fibrillation. Mackay, Mooslin, and Leeds[3] reported that a current greater than 1.5 amperes stopped fibrillation and in no instance

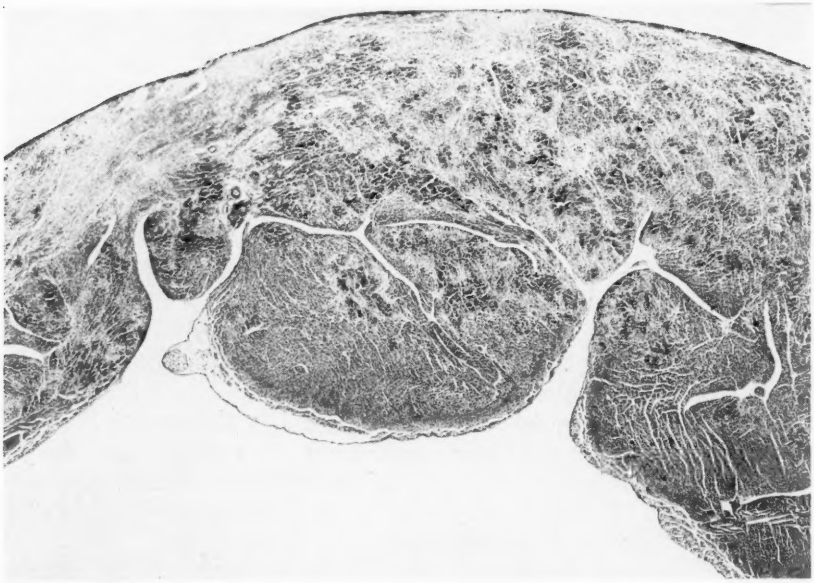

Fig. 1*A*.—Extensive necrosis and fibrosis of the right ventricle nine days postoperatively. The heart was defibrillated with a single shock of 230 volts at 1/10 second. (×13.)

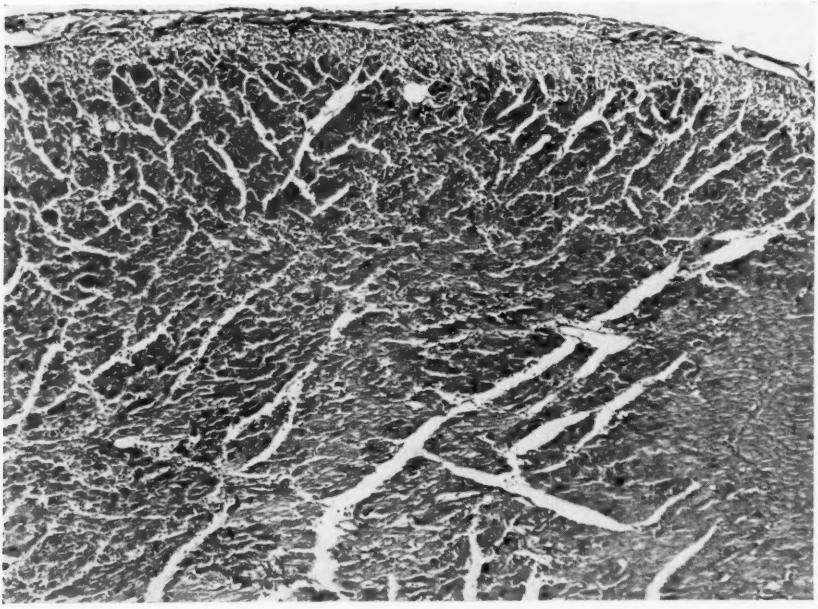

Fig. 1*B*.—Extensive necrosis and fibrosis of the myocardium of the left ventricle eight days postoperatively. The heart was defibrillated with a single shock of 230 volts at ¼ second. (×58.)

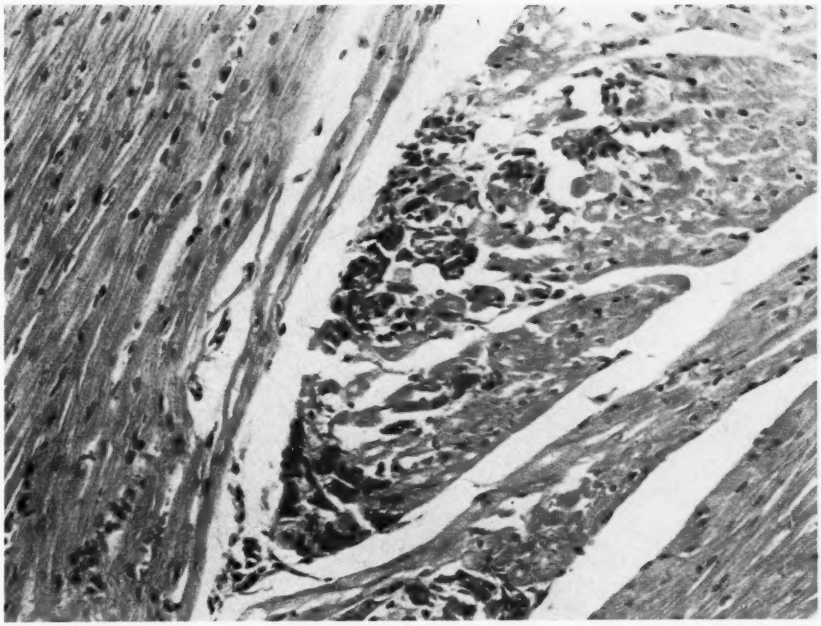

Fig. 1*C*.—Minimal focal necrosis of the myocardium of the left ventricle eight days postoperatively. The heart was defibrillated with a shock of 130 volts at 1/10 second. (×200.)

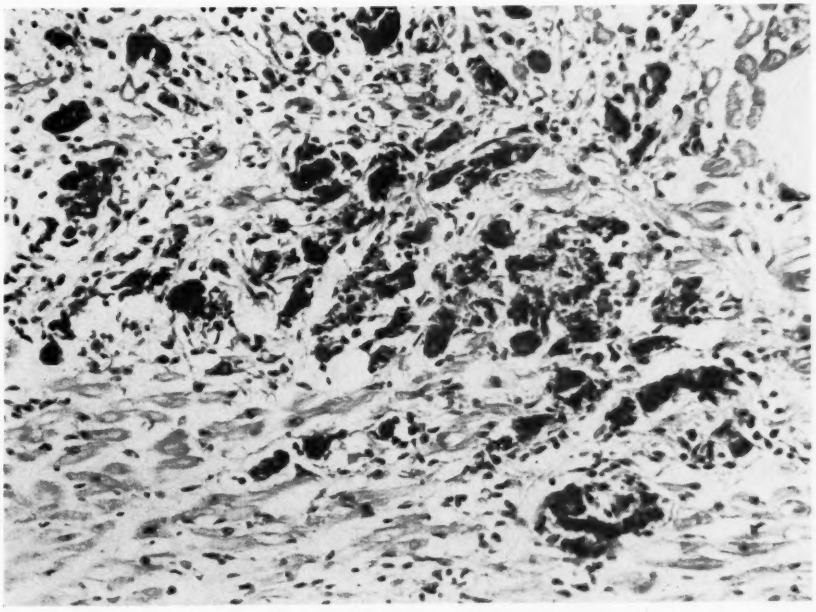

Fig. 1*D*.—Minimal lysis, necrosis, and fibrosis of the myocardium of the right ventricle twelve days postoperatively. The heart was defibrillated with a shock of 230 volts at 1/10 second. (×200.)

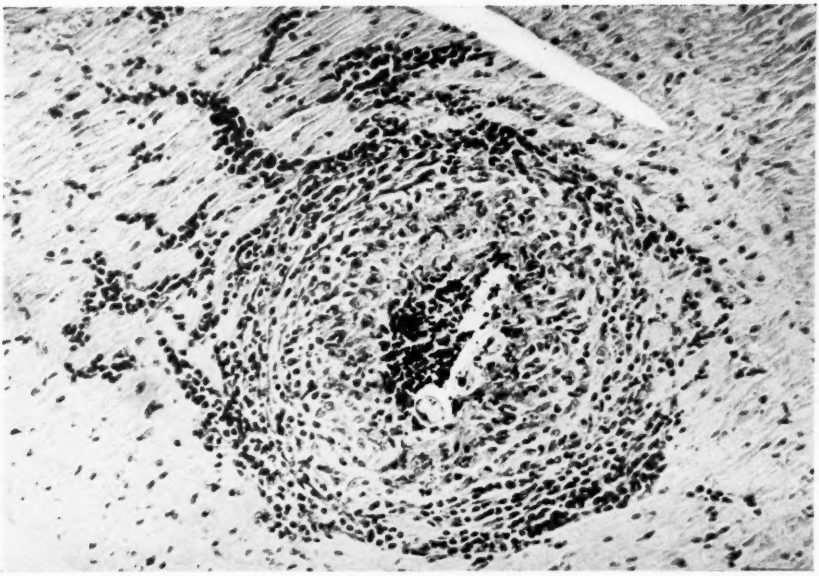

Fig. 1*E*.—Moderate necrosis and inflammation of a coronary artery of the left ventricle twenty-six days postoperatively. The heart was defibrillated with a shock of 230 volts at 1/10 second. (×200.)

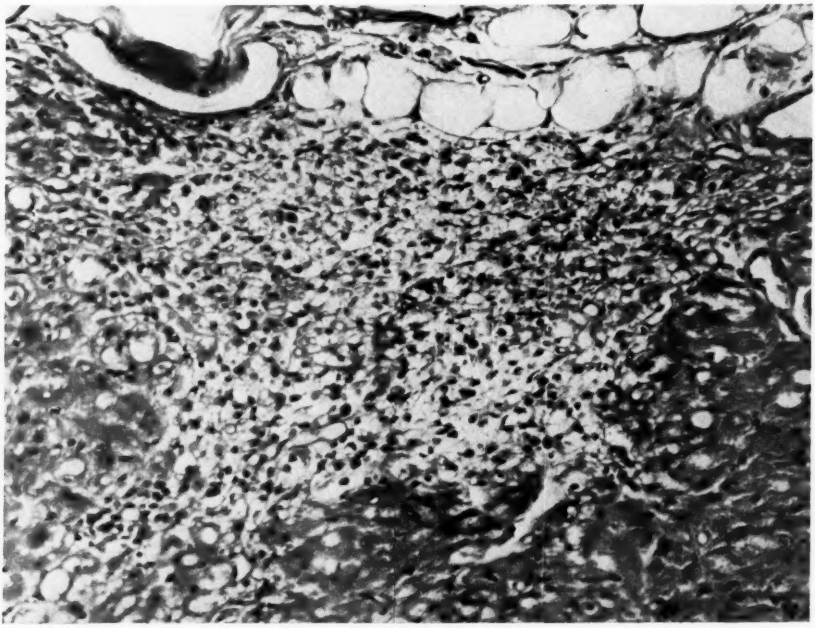

Fig. 1*F*.—Minimal focal subepicardial lysis and degeneration of the myocardium of the left ventricle fourteen days postoperatively. The heart was defibrillated with a shock of 230 volts at ¼ second. (×200.)

produced it. They concluded that currents of high amperage may be of either short or long duration to stop ventricular fibrillation and recommended 110 volts at 1/10 second.

Kirby, Johnson, Engelberg, and Rovis[1] compared the effectiveness of various voltages at 1/10 second duration. They stated that 220 volts at 1/10 second was uniformly more successful than lower voltages at 1/10 second in defibrillating canine hearts. They reported that this voltage at 1/10 second duration did not cause burns of the heart; but if the shock lasted ½ second there was a grossly visible burn. In the present series voltages and duration of shock were compared. Statistically there did not appear to be any significant difference in the ability to defibrillate the heart using the various voltages at the durations studied. There were myocardial burns in all four groups. However, there was an increased incidence of burns of the heart using 230 volts at ¼ second compared to the other groups. The burns of the hearts defibrillated with 130 volts were small and not visible grossly. The only severe burns occurred in the hearts defibrillated with 230 volts.

SUMMARY

Ventricular fibrillation was produced in 48 dogs. The hearts were allowed to fibrillate for four minutes and cardiac massage was then employed for two minutes. An attempt was then made to defibrillate the hearts with single electric shocks of various voltages and durations. There did not appear to be a significant difference in the ability to defibrillate the heart using 130 volts at 1/10 or ¼ second duration compared to 230 volts at 1/10 or ¼ second duration. However, there was a significant increase in number and severity of burns with 230 volts at ¼ second.

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EFFECTS OF EXTERNAL ELECTRICAL PACEMAKER STIMULI ON THE HUMAN HEART

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EXPERIMENTS in this laboratory have indicated that the rate of the heartbeat could be regulated externally by means of bioelectrical currents traversing the esophagus with sufficient intensity to excite the sino-auricular node and to elicit maximal cardiac response. Previously, Sweet[1] reported reactivation of the human heart by applying in the open chest an electrical current to the sino-auricular nodal area. Callaghan and Bigelow[2] have regulated the heart action of normal and hypothermic animals by electrical stimulation with an intracardiac electrode threaded through the venous system. In clinical practice, ventricular rhythm has been maintained and controlled by excitation currents from external electrodes applied to the closed chest wall.[3] The present investigation was designed to evaluate in greater detail the effect of electrical stimulation of the human heart from the standpoint of ectopic regulation of its rhythm under near normal conditions and during periods of operative stress.

METHOD OF STUDY

Twenty-six adult hospital patients, ranging in age from 24 to 85 years, volunteered to serve as subjects. Group I consisted of 16 subjects with normal sinus rhythm in whom electrical repetitive impulses were applied without preliminary sedation or other premedication. Group II consisted of 10 surgical patients who consented to electrical cardiac stimulation under Pentothal-spinal anesthesia during the course of elective surgical operations.

The electrical stimulator or pulse generator used in this study was specially constructed by the Mederaft Electronics Corporation. The principle of the Thyratron tube was utilized to provide stimulating impulses of approximately 1.36 amperes regulated for automatic delivery at predetermined specific rates. The amplitude of the output voltage from 1 to 100 volts, the pulse frequency from 20 to 120 cycles and the pulse width adjustable from 20 to 120 milliseconds were subject to selection and adjustment by manipulation of knobs on the panel of the instrument.

The active or stimulating electrode was constructed from a medium-sized stomach tube to which were affixed 5 coiled ring electrodes each about 1 cm. in diameter and equidistant from each other by ½ cm. These rings were connected to fine insulated wires which traversed the interior of the rubber

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tube and were attached to a base plate which in turn was connected to the stimulator. By means of the distributor at the base plate the current could be directed through one or more of the ring electrodes for the closest possible approximation to the sino-atrial node. The technique for placement of the esophageal electrode was that used for ordinary gastric intubation. An inactive or dispersive electrode was not applied to the chest wall. The frequency and other characteristics of the electrical pulses were routinely recorded on a two-channel direct writer simultaneously with peripheral arterial pressure

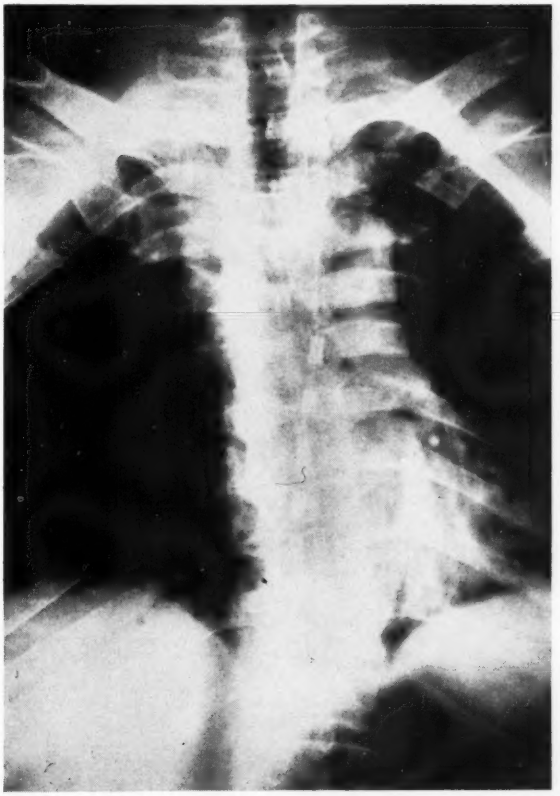

Fig. 1.—X-ray showing position of esophageal lead in patient.

tracings taken through a blood pressure cuff connected to a Sanborn electromanometer. These synchronous recordings of repetitive electrical pulses measured against systolic ejection peaks of the pressure tracings were used to evaluate the effectiveness of the stimulating impulses. Cardioscope visualization, kymoroentgenograms, and electrocardiograms in conjunction with the above were utilized whenever possible.

An experimental test in subjects of Group I consisted of cardiac stimulation by currents derived from output voltages increased in stages from 10 through 100 volts with variations of the pulse frequency and pulse width from the minimum to the maximum setting at each voltage level. In the subjects of Group II, the esophageal lead was introduced just after anesthesia was administered and the proper connections for recording the blood pressure and electrical impulses were made. The pacemaker was set at the beginning of the operation to deliver repetitive stimuli at frequencies greater than the initial heart rate and was interrupted only for brief periods to assess the heartbeat under its own rhythm.

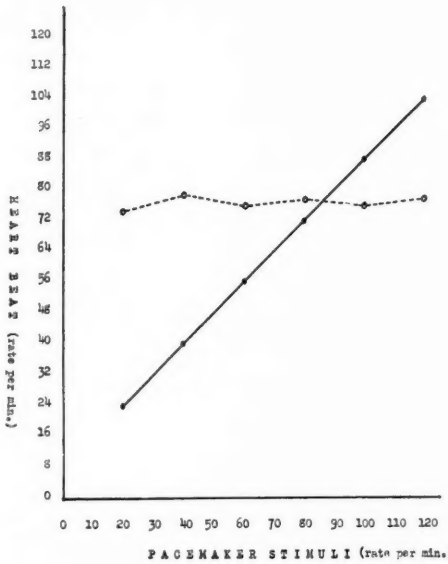

Fig. 2.—Shows no relationship of heart rate to pacemaker stimuli of 10 volts potential, pulse width of 20 milliseconds, and increasing frequency of stimuli from 20 to 120 per minute.

RESULTS

Group I: A total of 54 electrical tests of the heart through all tolerable stimulatory ranges were made in the 16 volunteers. Electrical impulses up to 10 volts with variations up to the maximum in frequency and in pulse width failed to increase the number of heartbeats. Within the 10- to 20-volt range, extrasystoles were occasionally detectable on the pressure pulse tracings. At the 20-volt level a relationship was noticeable between the number of ventricular contractions and the frequency of electrical impulses when such stimulating impulses exceeded 70 per minute. The correlation between heart rate and frequency of stimulation became more nearly equal as the voltage increased above 20 volts. In the 30- to 50-volt range with frequencies of 80 to 120 per

minute correspondence between the heart rate and number of impulses were exact in 90 per cent of the subjects. In the other 10 per cent, the same trend of correlation existed but with frequencies above 110 per minute the heart rate failed to follow suit. Abrupt change to effective stimulating currents with frequencies above the natural heart rate consistently reproduced the pattern of "take over" by the pacemaker. Increase of the pulse width from 10 to 70 milliseconds did not alter the response of the heartbeat but an electrical pulse width of 80 to 120 milliseconds even at low voltages caused pain referable to the chest over the site of the esophageal lead. Rhythmic contractions of the upper abdomen due to diaphragmatic stimulation were noted in about one third of the patients. These contractions followed the exact rhythm of the stimulator and were eliminated by adjustments on the base plate directing the current

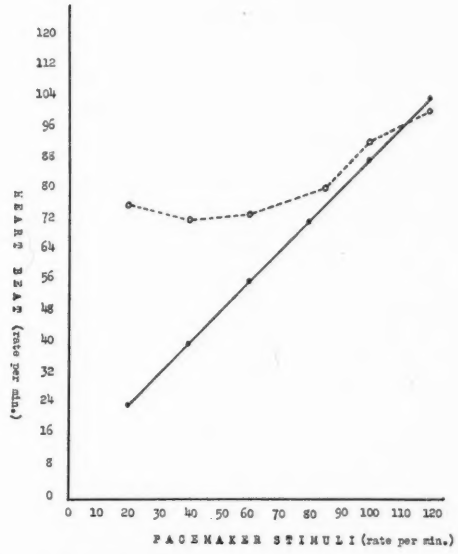

Fig. 3.—Shows a moderate increase in heartbeat but no correlation with pacemaker stimuli of 20 volts output, 20 milliseconds pulse width, and frequency of 20-120 impulses per minute.

of excitation through other electrodes. All the patients tolerated electrical impulses up to the 60-volt level but with higher voltage output pain in the chest was intolerable. Ventricular fibrillation due to electrical stimulation did not occur as a complication in this study and harmful clinical effects either immediate or delayed were not noted.

Group II: In all the 10 patients of this group the heartbeat was "taken over" by the pacemaker during the operative procedure for which they were admitted to the hospital. The electrical stimulations were repetitive at a frequency of 100 to 120 per minute with a pulse width of 30 milliseconds from a 40-volt output. Control of the heart rate commenced immediately after

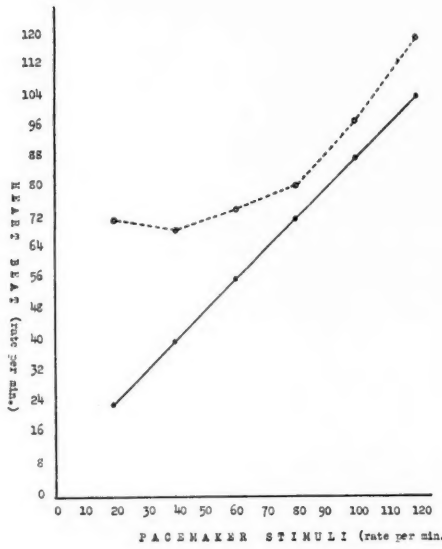

Fig. 4.—Shows correlation of heartbeat and pacemaker stimuli of 40 volts, pulse width 20 milliseconds and frequencies above 80 per minute. Stimuli less than 80 per minute did not affect the heart rate.

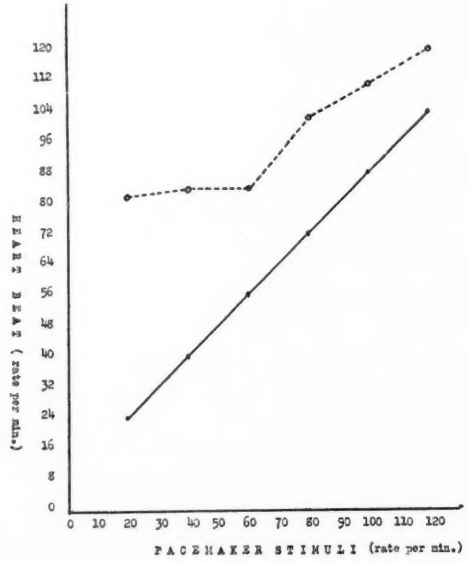

Fig. 5.—Shows almost exact correlation of heartbeat and pacemaker stimuli of 50 volts, pulse width 20 milliseconds and frequencies about 80 per minute. Stimuli delivered below the rate of 80 per minute did not affect the heart rate.

administration of anesthesia and external regulation at 100 to 120 beats per minute was continued throughout the entire operative period which averaged 75 minutes. The preanesthetic heart rate averaged 80 per minute and the postoperative rate, after discontinuation of the pacemaker, averaged 94 beats per minute. The operations performed under pacemaker control were limited to appendectomies and herniorrhaphies. Reversion to normal sinus rhythm was immediate in every case. During the period of stimulation the mean blood pressure as denoted by the pressure tracings showed no severe fluctuations or depressions and persisted at a steady level throughout the stimulatory period. Seven patients noted no adverse effects after recovery from the prolonged stimulation under anesthesia and surgery. Three patients complained of a dull ache in the chest which subsided within seventy-two hours.

DISCUSSION

Electromanometric tracings of the pulse form of the brachial artery established proof of correlation between external stimulation of the heart and ventricular contractions and reflected changes in the peripheral blood pressure during the period of myocardial stimulation. Kymoroentgenograms revealed the increased frequency of auricular and ventricular beats elicited by the pacemaker and showed these to follow in orderly sequence. Electrocardiograms taken at the beginning and end of electrical stimulation of the heart showed no pathologic changes due to prolonged stimulation.

The external pacemaker has been utilized as an effective means of resuscitation of the heart in ventricular standstill but has not been employed previously as a pacemaker to regulate heart action during surgery. On the basis of the present study the latter function is entirely feasible. Complete "take over" of the heart rate throughout an operation can help maintain efficient myocardial contractions and protection against vagal asystole in the poor-risk patient. Until a spark-proof electrode is developed, the procedure, as presented here, must be restricted to patients under spinal or intravenous anesthesia. The site for most effective stimulation of the heart has not been definitely ascertained. The sino-auricular node, the auricular ventricular junction, and the interventricular septum have been proposed, respectively, for optimal stimulation. In patients with normal sinus rhythm, repetitive electrical stimulation in the region of the sino-atrial node would appear to be the most logical site. Except when the conduction system and the cardiac musculature has been damaged organically, as by anoxia, stimulation at this site has generally caused complete myocardial contraction responses and normal-pattern electrocardiograms.

Wide variations have been reported in the literature on the values of stimulating electrical currents. For optimum defibrillation of the canine heart, Mackay[4] recommended electrical stimuli of 85 to 170 volts and 1.5 to 3.8 amperes. Zoll[3] used simple monophasic currents of rectangular shape and low voltage for resuscitation of the human heart in standstill. Hopp and Bigelow[5] obtained effective stimulation of the heart with currents from 0.01 to 0.1 ampere while Herrod and his associates[6] employed pacemaking currents

up to 3 amperes. The characteristics (voltage, amperage, etc.) of pacemaker impulses have not been standardized and the variable values noted in the literature can be accounted for by the type of electrical impulse generated, the method of application of the stimulus through the open or closed chest, the resistance of the heart, and other bioelectric effects.

SUMMARY

1. Physiologic effects of an electric stimulator on hearts with normal sinus rhythm were studied. Efficient myocardial contractions at a rate greater than the normal sinus rhythm can be produced by means of repetitive electrical stimulation.

2. The heart rate of 16 unanesthetized subjects and 10 anesthetized patients undergoing surgery was controlled externally by means of an electric pacemaker.

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SOLITARY BENIGN NEUROFIBROMA OF THE LUNG COEXISTENT WITH PULMONARY TUBERCULOSIS

Report of a Case with Review of the Literature

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WHILE intrathoracic neurogenic tumors are fairly common, primary neurogenic tumors, arising and confined within the pulmonary substance, are quite unusual. This report is concerned with a case of benign intrapulmonary neurofibroma associated with active pulmonary tuberculosis.

In 1940, Rubin and Aronson[1] reported finding at autopsy multiple tumor nodules in and on the surfaces of both lungs. These tumors proved to be neurofibromata and no evidence of generalized von Recklinghausen's disease could be found. Bartlett and Adams,[2] in 1946, reported one case of a neurinoma, benign, primary and solitary at the hilum of the left lung. It was firmly attached to the left bronchus, distal to the main bifurcation. A pneumonectomy was performed and an uneventful course followed. There was no evidence of recurrence at the time of writing. Touroff and Sapin,[3] in 1949, reported the surgical removal of a solitary neurofibroma from the apex of the lower lobe of the right lung. It was well encapsulated and readily removed from the lung parenchyma. Diveley and Daniel,[4] in 1951, reported 2 cases of neurogenic tumor, primary in the lung. The first was a neurofibroma, circumscribed, hard and benign, found in the middle lobe of the right lung. The middle lobe was resected and no evidence of recurrence was evident at the time of their report. Their second case was a neurogenic sarcoma in the upper lobe of the left lung, treated by pneumonectomy. This patient died four months after surgery, presumably due to metastases. Lane, Murray, and Fraser,[5] in 1953, described a neurilemmoma of the lung confirmed by tissue culture. This tumor, so termed by A. P. Stout, is thought to arise from the nerve sheath of Schwann and is not to be confused with neurofibroma. However, the case described was an example of a solitary, benign, neurogenic tumor arising within the substance of the lung.

CASE REPORT

S. M., a 33-year-old white woman, was admitted to the Pittsburgh Tuberculosis Hospital on Oct. 14, 1954, because of pulmonary tuberculosis detected on routine chest x-ray taken because her brother was undergoing treatment for this condition. The x-ray examination revealed scattering infiltrative disease in the upper portion of the left lung field and haziness

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high in the apex of the right lung. Blood counts, blood chemistry, blood serology, and urinalysis at regular intervals remained within the normal range. Bronchoscopy had revealed tuberculous endobronchitis of the left upper lobe bronchus. In January, she was operated on for acute appendicitis and recovered without complication. Treatment of the tuberculosis consisted of chemotherapy and routine sanatorium care. The sputum on smear and culture converted to and remained negative by February, 1954, only four months after admission. Bronchoscopy in June, 1954, revealed the left upper lobe endobronchial tuberculosis to be markedly improved. Chest x-rays were taken at appropriate intervals after admission and the lesion in the left lung showed progressive improvement until it appeared quite stable and under control. However, the lesion in the right apex, first described as a haziness and then as a linear density in the outer zone of the second interspace, gradually increased and became circumscribed and was finally described as lemon-sized. Planography on Aug. 13, 1954, showed it to be a solid, noncavitating lesion best delineated well to the posterior (Figs. 1 and 2). At this time, it was felt that this might represent a neoplastic rather than a tuberculous lesion. Bronchoscopic re-examination was performed and the left upper lobe bronchial lesion was found to be well healed and no abnormality of the right upper bronchus was seen. Bronchoscopic washings from the right upper lobe were reported negative for both tubercle bacilli and tumor cells. Other x-ray and fluoroscopic examinations were performed and the consensus of medical and surgical opinions was that surgical exploration was indicated and that the most likely diagnosis was a neurogenic tumor of the chest wall. This was based on the circumscribed appearance of the lesion and its apparent position against the posterior chest wall on the lateral aspect of the vertebral column.

Operation.—On Sept. 2, 1954, in the presence of an arrested, contralateral, left upper lobe tuberculous lesion, a right thoracotomy was performed through the bed of the resected fourth rib. A few filmy adhesions over the apex of the upper lobe were divided and the lesion was found to lie in the posterior apical portion of the upper lobe rather than on the chest wall. It was well rounded and approximately the size of a small lemon. It felt soft and almost cystic, similar to a dermoid or inspissated tuberculous lesion, rather than hard or nodular. At its base, near the segmental bronchus, some nodularity was palpable. It was thought that this was a tuberculous lesion and, since it was definitely localized, segmental resection was decided upon. The apical bronchus was isolated and dissected and a partial stenosis was noted a short distance (1 cm.) from its origin, just at the margin of the mass. A second small bronchus, arising immediately medial to that of the apical segmental bronchus, was found to be involved and hence was included in the resection. This probably represented either an anomalous or an accessory apical bronchus, since both the anterior and posterior segmental bronchi were positively identified. The resection proceeded without difficulty and, at the conclusion, the anterior and posterior segments of the upper lobe and the middle and lower lobes expanded readily without evidence of air leak or blood loss.

Pathologic Report.—The specimen consisted of the apical segment of the upper lobe of the right lung and a small fragment of bronchial stump. The segment of the lung was collapsed and contained in the apex a rounded, thickened, yellowish nodular area, 2 cm. in diameter. The pleura over this area was grossly thickened but intact. Beneath this white nodular area, there was a small area, 1 cm. in diameter, of necrotic lung tissue containing much blood and fragmented tissue. Also included was a small fragment of bronchial stump which consisted of an ellipse of bronchial tissue curled upon itself. On microscopic examination, sections of the lung revealed a solid neoplastic process located within the parenchyma and partially covered by pleura. The tumor was composed of elongated fusiform fibroblastic cells which in some areas produced small amounts of collagen. The cells were well differentiated and showed no pleomorphism and infrequent mitotic figures. The tumor was moderately vascular and contained irregular thin-walled sinusoidal channels, for the

Fig. 1.

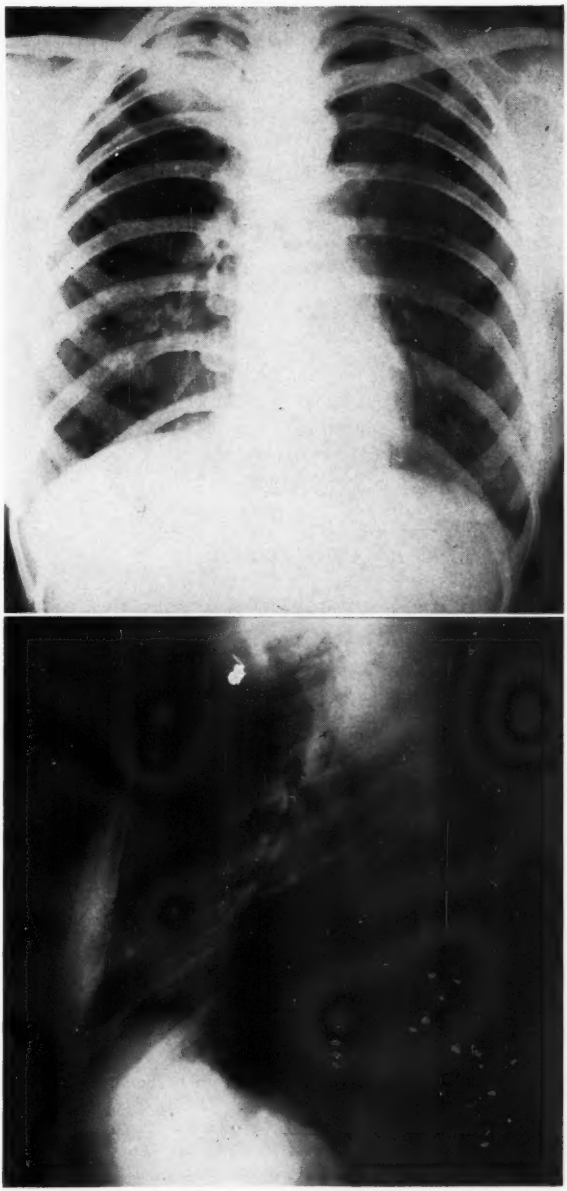

Fig. 2.

Fig. 1.—Posteroanterior projection nine days preoperatively, showing a circumscribed opacity in the apex of the right upper lobe.

Fig. 2.—Lateral projection made at the same time which reveals the posterior postion of the lesion.

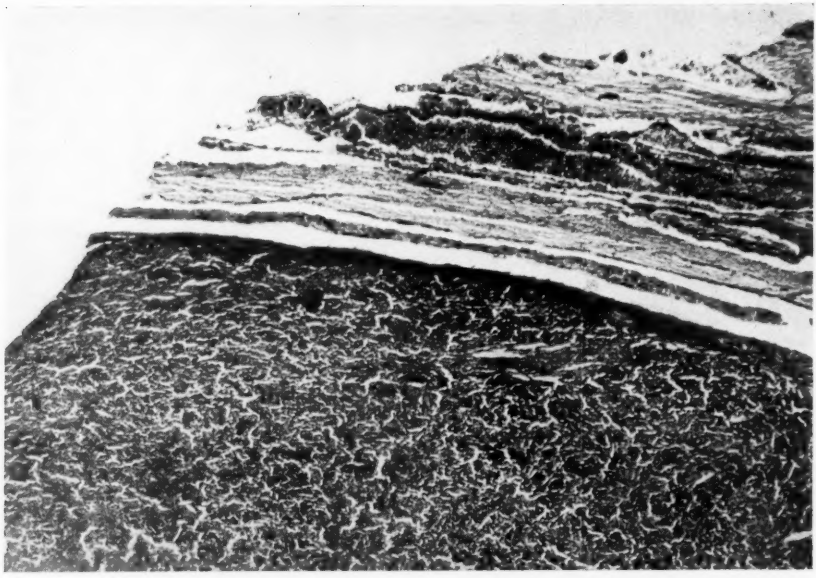

Fig. 3.—The dense cellularity of the tumor occupying the lower half of the illustration is evident. The linear area of dark cells above the capsule of the tumor represents blood within a compressed bronchus. (Low-power photomicrograph.)

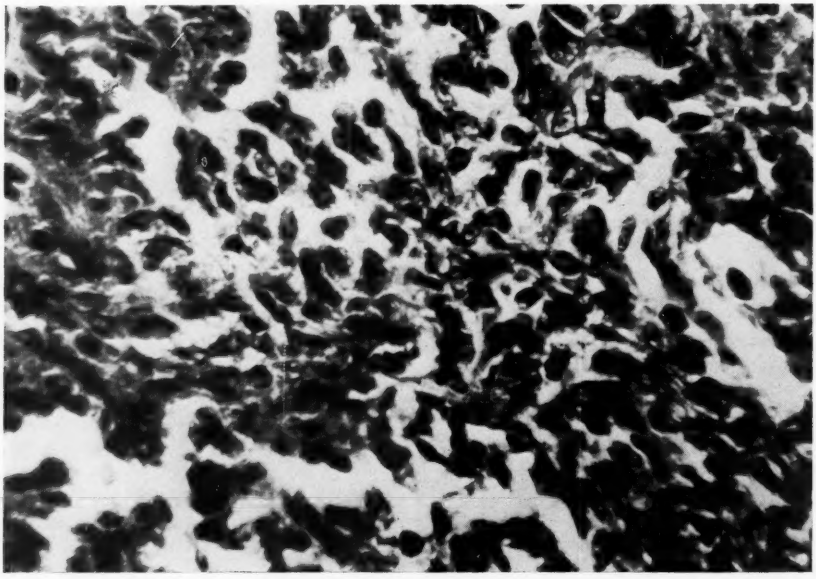

Fig. 4.—Elongated fusiform fibroblastic cells form the entire tumor. (High-power photomicrograph.)

most part, empty of blood. Areas of interstitial edema, interstitial hemorrhage, and early liquefaction were observed. There was no evidence of a tuberculous inflammatory reaction within the adjacent parenchyma. A section of the bronchial stump revealed no abnormalities.

Diagnosis.—Neurofibroma of the lung (Figs. 3 and 4).

Postoperative Course.—The postoperative course was uneventful. Re-expansion of the remainder of the lung was complete within forty-eight hours and the wound healed per primum (Fig. 5). There was no evidence of activation of tuberculosis, but she continued on the antituberculous regime in the hospital until Nov. 6, 1954, because of this possibility. No evidence of recurrence has been found twenty-one months later and the patient has remained well in all respects.

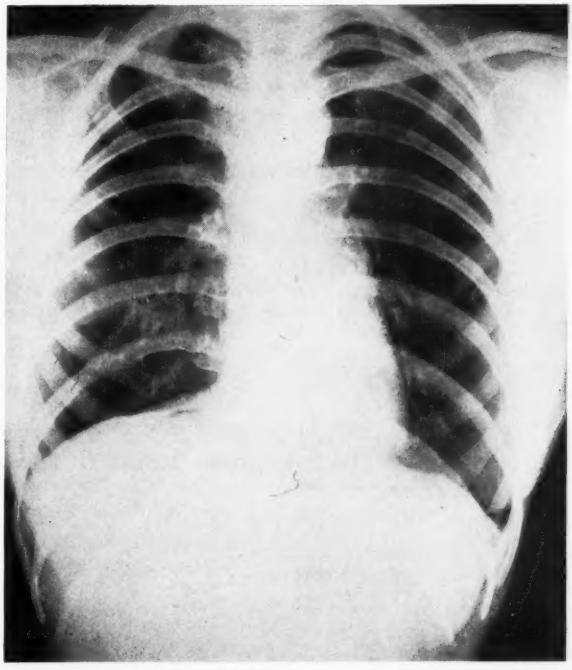

Fig. 5.—A projection made about eight weeks following surgery, which shows complete re-expansion of lung with minimal pleural reaction.

COMMENT

From the paucity of the literature and the cases reported therein, it is apparent that any neurogenic tumor arising within the lung substance is unusual, and one which is benign and solitary is still more unusual. Of those reported in the literature, one was described as a neurinoma, one as a neurilemmoma, and two as neurofibromata. A third neurofibroma, or the fifth benign neurogenic tumor fulfilling these requirements, is herewith reported. This tumor is notable in view of the fact that it was discovered and seemed to increase in size while the patient was undergoing treatment for active pulmonary tuberculosis in the contralateral lung. It has been reported that the incidence

of bronchogenic carcinoma in autopsied cases of active pulmonary tuberculosis is about 1.4 per cent.[6] However, the association of solitary tumors with pulmonary tuberculosis is quite rare and, since such lesions may well be malignant, surgical exploration and resection to the extent indicated by the nature of the lesion is the treatment of choice. The possibility of associated serious lesions must be kept in mind during the treatment of all tuberculous patients to prevent such lesions from reaching a stage of inoperability before they are recognized.

SUMMARY

Benign primary intrapulmonary tumors of neurogenic origin are rare within the lung, although similar tumors are found fairly often extrapleurally within the thorax. A case of neurofibroma of the right upper lobe, associated with active pulmonary tuberculosis, is reported. This appears to be the fifth such tumor to be described and the first in which there was an associated pulmonary tuberculosis present. It was of interest that this tumor was seen to expand in size while the patient was undergoing medical treatment for active pulmonary tuberculosis.

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A BRONCHOSCOPIC TABLE DESIGNED FOR OFFICE USE

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GARDEN CITY, N. Y.

THE current trend toward outpatient and office bronchoscopy has necessitated many modifications of equipment, depending upon individual taste and facilities. Three years' experience with office endoscopy has influenced me toward a gradual change in concept of a well-functioning bronchoscopy unit with particular emphasis on efficiency and convenience. The necessity for conserving space and personnel has led to progressive concentration of equipment. This has eventuated in the construction of a single unit. This is essentially a converted office examination table, modified, to incorporate the use of a motor-driven headrest, reflector light, power spray, suction system, battery system, and instrument receptacle for bronchoscopic implements. Little or no assistance is needed during the operation and very little space is required. This unit has functioned so smoothly that it appeared worthy of publication.

In Fig. 1, the small size of the table, identical to preconversion size, is demonstrated. It is not conspicuous as a special operating table and can be used for ordinary office examinations. As noted from this view, the headboard has been taken apart, sawed across, and reattached with hinges and reupholstered. The stirrups have been removed. In the left stirrup holder, an adjustable light has been built in for use with the head mirror, when administering anesthesia. Through the right stirrup holder slides a power-spray line, the tip of which prevents it from falling through. When administering anesthesia this is attached to a power-spray bottle. When not in use the line falls back by gravity. It just clears the floor in neutral position.

The patient sits upright on the table during anesthesia. The armboard has been modified by putting a hole in it at an oblique angle. Through this opening a megaphone-like receptacle can be inserted to hold the instruments during bronchoscopy.

In Fig. 2, the headrest and controls are seen as a close-up. The megaphone receptacle is in position. The two paired objects built into the side of the table are battery light controls, each with its own red pilot light to prevent undetected battery wastage when not in use. Adjacent to each pilot light is an off-on switch-rheostat combination. These have a wide range of selectivity, obviating any sudden increase in power which may cause burning out of the bulb. They are situated for convenience within easy reach of the operator.

From the Division of Thoracic Surgery, Department of Surgery, State University of New York College of Medicine at New York, and Department of Surgery, Nassau Hospital, Mineola, N. Y.

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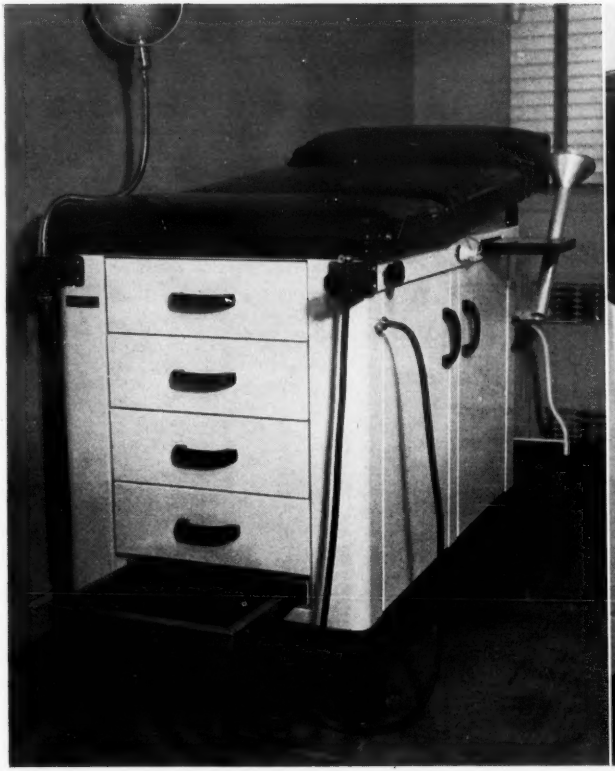

Fig. 1.

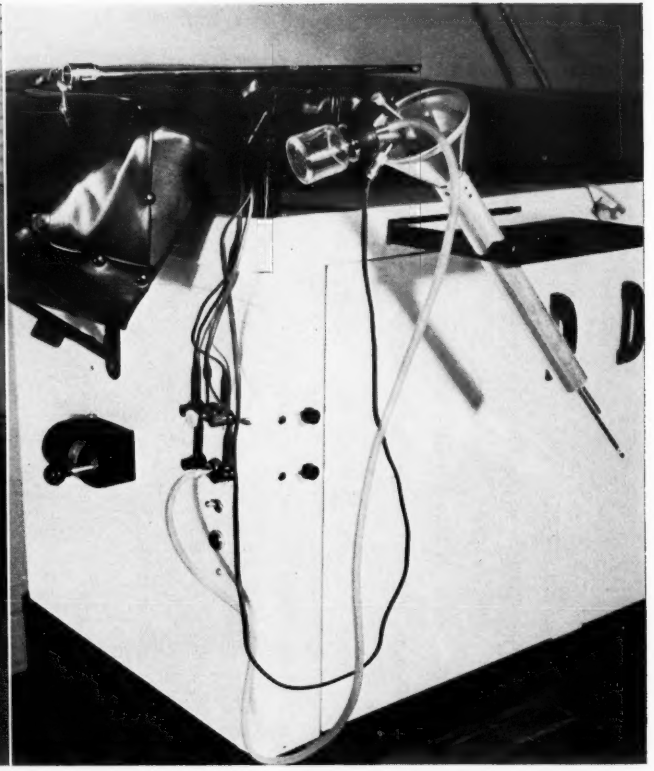

Fig. 2.

Fig. 1.—Rear view of bronchoscopy table. Uppermost drawer pulls forward to supply extra table length. See text.

Fig. 2.—Head view of bronchoscopy table. This view demonstrates headrest, motor control lever, battery terminals, suction and blower tips, single main switch with pilot light, and twin battery controls with individual pilots. Note also megaphone-receptacle in position, containing right-angle telescope and suction tip with built-in collecting jar used as handle.

The motor control for the headrest is situated directly beneath the headrest within easy reach of the operator. In the horizontal plane the lever is in neutral position; when raised upward the headrest is driven upward; when deflected downward the headrest is lowered. The motor can be stopped at any time in either direction by simply returning the lever to the neutral position. Headrest position can be varied from 60 degrees in the upward extreme to 90 degrees in the lowered position.

It is of additional interest that motor control can be effected by either knee action or finger operation. This facilitates head raising or lowering by the operator while looking through the bronchoscope without the sudden or jolting motions often experienced with mechanical systems. Another important feature is the utilization of limit switches, applied to the rack, which moves the headrest. These automatically trip the power when the headrest reaches either extreme position obviating the necessity to manually stop the motor when these danger points are reached.

To the right of the motor control are four built-in battery terminals. Usually the bronchoscope is powered by the uppermost pair and a telescope from the lower. Between the two upper terminals is a blower tip with a removable tight rubber cover when not in operation. This was designed for use with the Broyle bronchoscope but I do not use it.

Between the lowermost battery terminal is the suction spout which is connected by tubing to the suction tip through a two-hole rubber stopper in a specimen jar. This is used as a handle to manipulate the suction tip. While in the megaphone-receptacle, the weight of the jar prevents rotation and sucking up of its contents, although the jar can be emptied at any time by simply turning it upside down. When bronchial washings are taken, they are sent to the laboratory in the same jar which is easily detached and replaced by a clean one. Likewise, separate washings can be taken from both sides.

It should be noted that all controls and operations are within immediate and convenient reach of the operator. Incidentally, this table was designed for a right-handed surgeon but could be used by a left-handed person since the controls are neither delicate nor sensitive.

The advantages of the megaphone-like receptacle are obvious. The suction tip or telescope can be reached for and returned quickly and smoothly without assistance and without having to turn around to reach for them.

It is apparent during bronchoscopy that there is no confusion of wires or tubing from table to table or in front of the operator. No other pieces of equipment are seen except the table. Assistance is usually unnecessary. The sum total of these advantages is simplicity which is readily appreciated by the endoscopist. It is believed that many of the advantages of the table would still obtain in the operating room.

In Fig. 3, the interior of the table is seen. The cabinet door is open. The former cabinet space has been stripped of partitions and former backrest. In

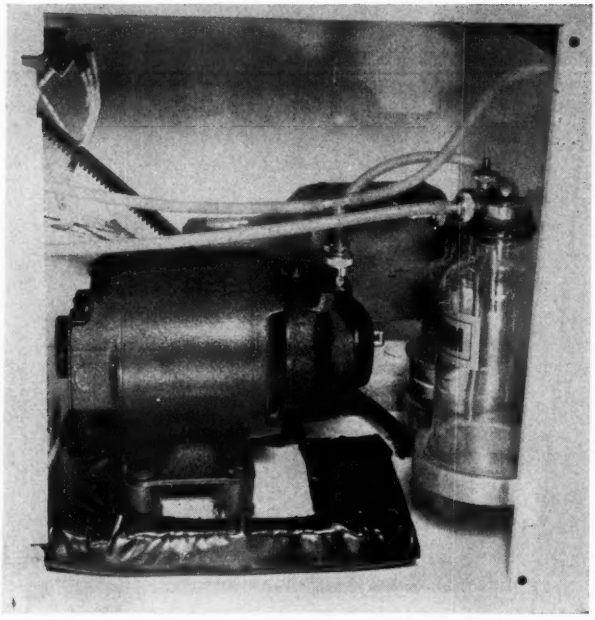

Fig. 3.—Interior of bronchoscopy table. See text.

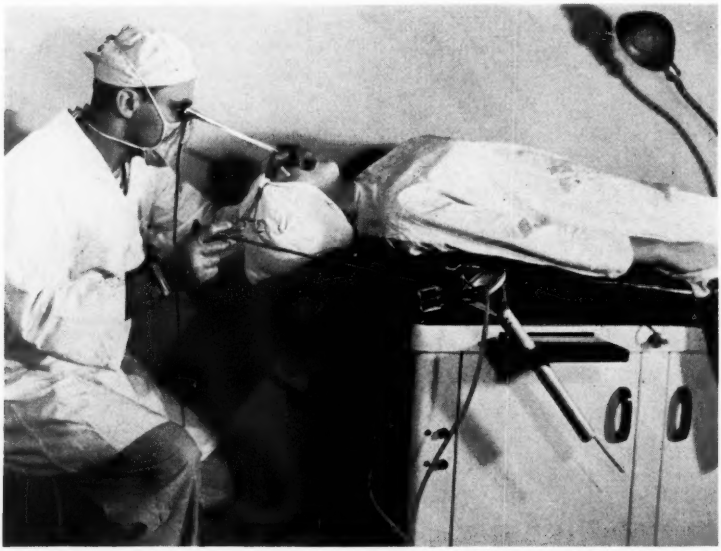

Fig. 4.—Bronchoscopy table in operation. Note controls, megaphone-receptacle with suction tip and built-in sputum jar handle. Note motor control in neutral position. Bronchoscope and right-angle telescope wired and in use.

the foreground is a suction-blower with appropriate bottles. A blower outlet bifurcates; one going to the power spray line at the rear of the table, and one to the control panel.

The suction tube extends only to control panel. Batteries are on the left and almost hidden from view. The suction motor is easily removed. To prevent vibrations, the suction motor is heavily padded with sponge rubber. In the background is a 1/12 horsepower motor with built-in gear box. The shaft is supported by a single brace from the bottom of the table. On the shaft is mounted a single gear which moves the headrest rack one inch per second, thus, only one external gear is necessary (hidden by suction motor).

Note the elevation limit switch mounted above the rack to trip power at the limit of elevation. A similar switch, which is hidden by the suction motor, limits depression.

In Fig. 4, the machine is in operation.

COMMENT

It is not the purpose of this paper to recommend office bronchoscopy in preference to operating room bronchoscopy. It may be of interest to state that only a few mild reactions to anesthesia have been encountered. Although accidents may occur anywhere, avoidance of poor-risk patients, those with allergic histories, and the use of ½ per cent pontocaine in small amounts has minimized the possibility of reaction. An emergency medication—cardiac arrest pack is kept inside the table but fortunately has never been used.

Patients are admitted to the office, without breakfast, one hour prior to bronchoscopy at which time they are sedated with morphine-atropine and Nembutal. Following bronchoscopy, they are afforded a rest period of one hour after which they are permitted to leave the office. We have found that both the patients and their referring physicians are highly in favor of this as an office procedure.

SUMMARY AND CONCLUSIONS

1. A bronchoscopy table has been designed for office use which has the advantages of motor-driven headrest, built-in flexible light, built-in battery power source, built-in suction power spray unit, and a built-in megaphone-receptacle for instruments.

2. Little or no assistance is required during bronchoscopy due to the advantages afforded by the table.

3. Complete bronchoscopy can be performed with very small space requirement.

4. Office bronchoscopy can be performed with minimal risk and great advantage to both the patient and his physician.

Evarts A. Graham

The Editorial Board and Publishers of the JOURNAL OF THORACIC SURGERY announce with deep sorrow the death on March 4 of Dr. Evarts A. Graham, Editor of the JOURNAL since its inception in 1931.

Dr. Graham's guidance was felt in every matter which concerned the JOURNAL. His responsibilities and interests, many of them world-wide, did not preclude his careful reading of manuscripts and his wise and kind direction even in details of editing and publication.

An obituary will be published in an early issue of the JOURNAL.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

THIRTY-SEVENTH ANNUAL MEETING
MAY 4, 6, AND 7, 1957
PALMER HOUSE, CHICAGO, ILL.

Program

MEMORANDA FOR GUIDANCE OF ESSAYISTS AND DISCUSSANTS

1. In accordance with the By-Laws of the Association the papers which are read at the Meeting shall be given to the Reporter *immediately after presentation.* If these papers are not submitted promptly their publication may be delayed.
2. In publication it is customary to group discussions together on a series of papers. Any delay in the return of corrected discussions to the Editor means that publication of all papers on the subject will be held up. This is manifestly unfair to those who are conscientious in the prompt submission of their remarks for publication. Undue tardiness may mean that the discussion will not be published.
3. *Regular Program.*—Essayists (except in symposia) will be limited to 15 minutes for presentation.
4. *Thoracic Surgery Forum.*—Essayists will be limited to 10 minutes for their presentations. Papers on the Thoracic Surgery Forum as submitted for publication will be limited to 3,000 words. At the discretion of the President and within the limits of the allotted time, one or more discussions per paper will be allowed following all formal presentations.
5. *Discussants.*—Discussants will be limited to 3 minutes. Only members of the Association and invited speakers will have the privilege of discussing papers.

MOTION PICTURE SESSION

Motion pictures will be shown on May 4, 5, 6, and 7. Please consult official program for titles.

Saturday Morning, May 4, 1957

8:30 A.M. Business Meeting.

9:00 A.M. Presentation of Presidential Badge and Chain of Office by the *Society of Thoracic Surgeons of Great Britain and Ireland.*
Scientific Session: REGULAR PROGRAM.

1. **A Roentgen Study of the Evolution of Carcinoma of the Lung.** Leo G. Rigler, Minneapolis, Minn.
2. **Intraepithelial Carcinoma in the Tracheobronchial Tree of Lung Cancer Cases.** Oscar Auerbach, J. Brewster Gere (by invitation), Joseph M. Pawlowski (by invitation), and Arthur Purdy Stout (by invitation), East Orange, N. J.
3. **Bilateral Therapy for Unilateral Spontaneous Pneumothorax.** Ivan D. Baronofsky, Horace G. Warden (by invitation), James L. Kaufman (by invitation), Joseph Whatley (by invitation), and Joseph M. Hanner (by invitation), San Diego, Calif.
4. **The Surgical Treatment of Chronic Progressive Pulmonary Histoplasmosis.** John W. Polk (by invitation), Jose Cubiles (by invitation), Mt. Vernon, Mo., and W. W. Buckingham, Kansas City, Mo.

5. **A Study of the Role of Drug Resistance in the Surgical Treatment of Pulmonary Tuberculosis (Period January 1947—June 1956).** William R. Sweetman (by invitation) and John M. Salyer, Denver, Colo.
6. **The Value of Routine Pulmonary Function Studies in Thoracic Surgical Cases.** Hurst B. Hatch, Jr. (by invitation), Joseph K. Bradford (by invitation), and Alton Ochsner, New Orleans, La.

Saturday Afternoon, May 4, 1957

2:00 P.M. Scientific Session: REGULAR PROGRAM.

7. **Rigid Plastic Prostheses in Vascular Surgery.** F. X. Byron, Josh Fields (by invitation), Augustus Foster (by invitation), and Richard Hood (by invitation), Beverly Hills, Calif.
8. **Diagnostic and Physiologic Measurements Using Left Heart Catheterization.** W. S. Blakemore (by invitation), T. G. Schnabel (by invitation), P. T. Kuo (by invitation), and H. B. Conn (by invitation), Philadelphia, Pa.
9. **Effect of Exercise on the Diastolic Atrio-Ventricular Gradient in Mitral Stenosis.** Robert S. Litwak (by invitation), Philip Samet (by invitation), W. H. Bernstein (by invitation), Leonard Silverman (by invitation), Hyman Turkewitz (by invitation), and Milton E. Lesser (by invitation), Miami, Fla.
10. **Surgical Treatment of Transposition of the Aorta and Pulmonary Artery.** Thomas G. Baffes (by invitation), William L. Riker (by invitation), Arthur DeBoer (by invitation), and Willis J. Potts, Chicago, Ill.
11. **The Use of a Mechanical By-Pass During Cross-Clamping of the Aorta.** Harold King (by invitation), and Harris B. Shumacker, Jr., Indianapolis, Ind.
12. **Elective Cardiac Arrest: An Adjunct to Open Heart Surgery.** Donald B. Effler, Laurence K. Groves (by invitation), Harold F. Knight (by invitation), Wilhelm J. Kolff (by invitation), and F. Mason Sones, Jr. (by invitation), Cleveland, Ohio.
13. **Experiences With the Use of Cardioplegia (Induced Cardiac Arrest) in the Repair of Interventricular Septal Defects.** Conrad R. Lam, Thomas Geoghegan (by invitation), Charles Sergeant (by invitation), and Edward Green (by invitation), Detroit, Mich.

6:30 P.M.—8:30 P.M. COCKTAIL PARTY. Informal. Palmer House.

Sunday, May 5, 1957

No Scientific Session

For Motion Picture Session consult official program.

Monday Morning, May 6, 1957

8:30 A.M. Scientific Session: THORACIC SURGERY FORUM.

14. **The Effects Produced by Various Types of Pump Oxygenators During Two-Hour Partial Perfusions in Dogs.** Marian E. Molthan (by invitation), Stanley Gianelli (by invitation), Richard J. Best (by invitation), James A. Dull (by invitation), and Charles K. Kirby, Philadelphia, Pa.
15. **Acid-Base Balance During Prolonged Cardio-Respiratory By-Pass.** Matthias Paneth, Traveling Fellow of the Association, 1956-57 (by invitation), London, England, M. Nazih Zuhdi (by invitation), and William Weirich (by invitation), Minneapolis, Minn.
16. **Coronary Perfusion as an Aid to Open Heart Surgery Under Hypothermia in Man.** J. V. Maloney, Jr. (by invitation), S. A. Marable (by invitation), and W. P. Longmire, Jr., Los Angeles, Calif.

17. **Alterations in Renal Hemodynamics During and Following Resection of the Thoracic Aorta for Aneurysm.** George C. Morris, Jr. (by invitation), Raymond R. Witt (by invitation), Denton A. Cooley, John M. Moyer (by invitation), and Michael E. De Bakey, Houston, Tex.
18. **Physiological and Pharmacological Studies of Collateral Pulmonary Flow.** David State (by invitation), Peter F. Salisbury (by invitation), and Peter Weil (by invitation), Los Angeles, Calif.
19. **An Experimental Appraisal of the Finney Pyloroplasty in the Prevention of Esophagitis Following the Heller Myotomy.** Paul W. Herron (by invitation), George I. Thomas (by invitation), and K. Alvin Merendino, Seattle, Wash.
20. **Esophageal Motility in Achalasia (Cardiospasm) After Treatment.** Brian Creamer (by invitation), Arthur M. Olsen, Colin B. Holman (by invitation), and Charles F. Code (by invitation), Rochester, Minn.
21. **Postoperative Sodium Excretion Following Administration of Hypertonic Sodium Chloride Solution.** Joseph L. Kovarik (by invitation), and John F. Laws (by invitation). Sponsored by Hiram T. Langston, Chicago, Ill.
22. **Anatomic and Pathologic Studies of the Thoracic Duct.** Harvey W. Kausel (by invitation), Thomas S. Reeve (by invitation), Arthur A. Stein (by invitation), Ralph D. Alley, and Allan Stranahan, Albany, N. Y.
23. **The Study of Ventricular Fibrillation by Threshold Determinations.** Norman E. Shumway (by invitation), John A. Johnson (by invitation), and Richard J. Stish (by invitation), Minneapolis, Minn. Sponsored by F. John Lewis, Chicago, Ill.
24. **Direct Surgical Procedures on the Coronary Arteries: Experimental Studies.** Ormond C. Julian, M. Lopez-Belio (by invitation), and Donald Moorehead (by invitation), Chicago, Ill.
25. **Ligation of the Internal Mammary Arteries as a Means of Increasing Blood Supply to the Myocardium.** Robert P. Glover, and Julio C. Davila (by invitation), Philadelphia, Pa.
26. **The Gradual Closure of Interatrial Defects.** Robert B. Benjamin (by invitation), Robert S. Flom (by invitation), St. Paul, Minn., and F. John Lewis, Chicago, Ill.
27. **Physiological Considerations of Intracardiac Pressures Following Closure of Atrial Septal Defects.** Henry T. Bahnson, and G. Rainey Williams (by invitation), Baltimore, Md.
28. **Transplantation of the Homologous Canine Heart.** Salem F. Sayegh (by invitation), Max Halley (by invitation), and Oscar Creech, Jr., New Orleans, La.

Monday Afternoon, May 6, 1957

2:00 P.M. Executive Session. (Limited to Active and Senior Members.)

3:00 P.M. Scientific Session: REGULAR PROGRAM.

Address of the President, Cameron Haight, Ann Arbor, Mich.

Some Observations on Esophageal Atresias and Fistulas of Congenital Origin.

29. **Intrapericardial Bronchogenic Cysts: Report of Two Cases and Probable Embryological Explanation.** C. Harwell Dabbs (by invitation), E. Converse Peirce, II (by invitation), Knoxville, Tenn., and Ralph Berg, Jr., Spokane, Wash.
30. **Primary Repair of Traumatic Rupture of the Thoracic Aorta.** Robert G. Pontius (by invitation), Boston, Mass., and Oscar Creech, Jr., New Orleans, La.

31. **The Electro-encephalogram in Patients Undergoing Open Intracardiac Surgery With the Aid of Extracorporeal Circulation.** Richard A. Theye (by invitation), Robert T. Patrick (by invitation), and John W. Kirklin, Rochester, Minn.

7:00 P.M. COCKTAILS, BANQUET, AND DANCING, Palmer House.

Attendance Limited to Members of the Association and their ladies, invited speakers and their ladies.
Dinner dress preferred.

Tuesday Morning, May 7, 1957

9:00 A.M. Scientific Session: REGULAR PROGRAM.

32. **Cine-Fluorography in the Evaluation of Neuromuscular Diseases of the Esophagus.** Frederick S. Cross, Earle B. Kay, and George F. Johnson (by invitation), Cleveland, Ohio.
33. **Esophageal Hiatus Hernia of the Diaphragm: An Analysis of Surgical Results.** George H. Humphreys II, Jose M. Ferrer, Jr. (by invitation), and Philip D. Wiedel (by invitation), New York, N. Y.
34. **An Operation for Hiatus Hernia With Short Esophagus.** J. Leigh Collis (by invitation), Birmingham, England.
35. **Traumatic Rupture of the Diaphragm—Clinical Manifestations and Surgical Treatment.** Gerard Desforges (by invitation), John W. Strieder, Joseph P. Lynch, and Irving M. Madoff, Boston, Mass.
36. **Reconstruction of the Esophagus With Segments of the Colon.** William E. Neville (by invitation), and George H. A. Clowes, Jr., Cleveland, Ohio.
37. **Primary Tumors of the Heart: A Surgical Problem.** J. Gordon Scannell, and Hermes C. Grillo (by invitation), Boston, Mass.

Tuesday Afternoon, May 7, 1957

2:00 P.M. Scientific Session: REGULAR PROGRAM.

38. **Ventriculoplasty for Cardiac Aneurysm.** C. P. Bailey, H. E. Bolton (by invitation), and R. A. Gilman (by invitation), Philadelphia, Pa.
39. **Visual Repair of Congenital Aortic Stenosis During Hypothermia.** Henry Swan, S. Gilbert Blount, Jr. (by invitation), and Robert H. Wilkinson (by invitation), Denver, Colo.
40. **Surgical Treatment of Stenotic or Regurgitant Lesions of the Mitral and Aortic Valves by Open Cardiotomy.** C. Walton Lillehei, Richard A. DeWall (by invitation), Vincent L. Gott (by invitation), and Richard L. Varco, Minneapolis, Minn.
41. **The Surgical Correction of Aortic Insufficiency.** Warren J. Taylor (by invitation), Wendell B. Thrower (by invitation), Harrison Black, and Dwight E. Harken, Boston, Mass.
42. **The Clinical and Physiological Criteria for the Surgical Correction of Mitral Regurgitation.** Julio C. Davila (by invitation), P. Jumbala (by invitation), Robert G. Trout (by invitation), and Robert P. Glover, Philadelphia, Pa.
43. **The Application of Hydraulic Principles to the Coronary Circulation.** Bernard L. Brofman (by invitation), and Claude S. Beck, Cleveland, Ohio.